Archives of Neurology and Psychiatry

VOLUME 52 SEPTEMBER 1944 NUMBER 3

COPYRIGHT, 1944, BY THE AMERICAN MEDICAL ASSOCIATION

RELATION OF ABNORMAL COLLECTIONS OF CELLS IN POSTERIOR MEDULLARY VELUM OF CEREBELLUM TO ORIGIN OF MEDULLOBLASTOMA

JOHN RAAF, M.D.
PORTLAND, ORE.
AND
JAMES W. KERNOHAN, M.D.
ROCHESTER, MINN.

The external granular layer of the cerebellum, which makes its appearance in early fetal life, contributes to the growth of the cerebellum and then disappears within twenty months after birth, is composed of cells which resemble the cells in the medulloblastoma. This similarity has led to the theory that the cells of the external granular layer do not disappear in certain regions as quickly as in others and that the persistence of some of these cells is the origin of the medulloblastoma. In another study (to be published elsewhere [1]) of the same material that forms the basis of this investigation, in which we gave especial attention to the disappearance of the external granular layer and the growth of the molecular and internal granular layers of the cerebellum, we made the following observations:

The rate of decrease in width of the external granular layer does not differ significantly in the regions of the cerebellum studied.

The external granular layer persists until the child is 18 to 20 months of age and does not persist appreciably longer in one region than in another.

From the fifth prenatal month until shortly before birth the width of the external granular layer decreases slowly, but during the first nine to ten months of postnatal life the decrease is rapid.

There is little increase in the width of the molecular layer prior to one month before birth, but from birth, or shortly before birth, until the eleventh or twelfth postnatal month, the width of this layer increases rapidly.

The internal granular layer in the portions of the cerebellum studied shows a rapid increase in width during the last four intrauterine months and the first four or five postnatal months.

The external granular layer and the molecular layer have uniform widths throughout the cerebellar cortex, but the width of the internal granular layer increases unevenly; thus the cerebellum is thrown into folds.

During the course of this study, abnormal collections of cells were frequently noted in the region of the posterior medullary velum, and our attention was subsequently devoted to these cells.

Other authors have seen collections of abnormal cells in the cerebellum. Meschede [2] encountered 3 such cerebellar anomalies, and Pfleger [3] observed them in 75 of 400 cerebellums, this number not including several in which collections were noted with the naked eye only and were not checked microscopically. The heterotopias which Pfleger observed occurred in young, as well as in old, persons and varied greatly in size. Sometimes the cell masses were present in one hemisphere only, but often they occurred in both hemispheres simultaneously and were situated between the cortex and the dentate nucleus. Microscopic examination disclosed a finely granular network of basal substance with an irregular infiltration of cells similar to those occurring in the cortex. These cells frequently were scattered helter-skelter. In addition, there

From the Department of Surgery, Medical School of the University of Oregon (Dr. Raaf) and the Section on Pathologic Anatomy, the Mayo Clinic (Dr. Kernohan).

Abridgment of part of the thesis submitted by Dr. Raaf, formerly a fellow in Neurosurgery at the Mayo Foundation, to the Faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Neurosurgery.

1. Raaf, J., and Kernohan, J. W.: A Study of the External Granular Layer in the Cerebellum: The Disappearance of the External Granular Layer and the Growth of the Molecular and Internal Granular Layers in the Cerebellum, Am. J. Anat., to be published.

2. Meschede, F.: Heterotopie grauer Hirnsubstanz im Markstamme der Hemisphären des kleinen Gehirns: Drei Beobachtungen, Virchows Arch. f. path. Anat. **56:** 82-96, 1872.

3. Pfleger, L.: Beobachtungen über Heterotopie grauer Substanz im Marke des Kleinhirns, Centralbl. f. d. med. Wissensch. **18:**468-470, 1880.

were small cells, like those of the granular layer. These small cells were intermingled with ganglion cells. The blood vessels around the heterotopic masses were usually increased in number.

mented on the persistence of germinal clusters in the first postnatal month. He expressed agreement with Wanke [5] that these cells were related to unripe glial elements and neuroblasts. Winkelman and Eckel [6] stated that the intricacies

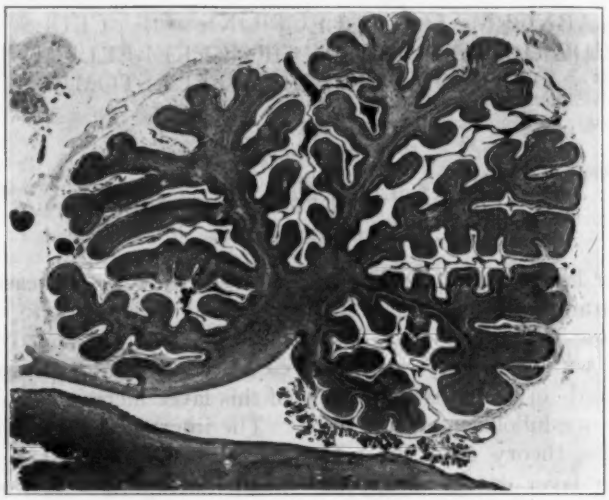


Fig. 1.—Cerebellum of a fetus of 7 months. The germinal bud is in the posterior medullary velum but is less prominent than that in younger fetuses. Hematoxylin and eosin stain; × about 6.5.

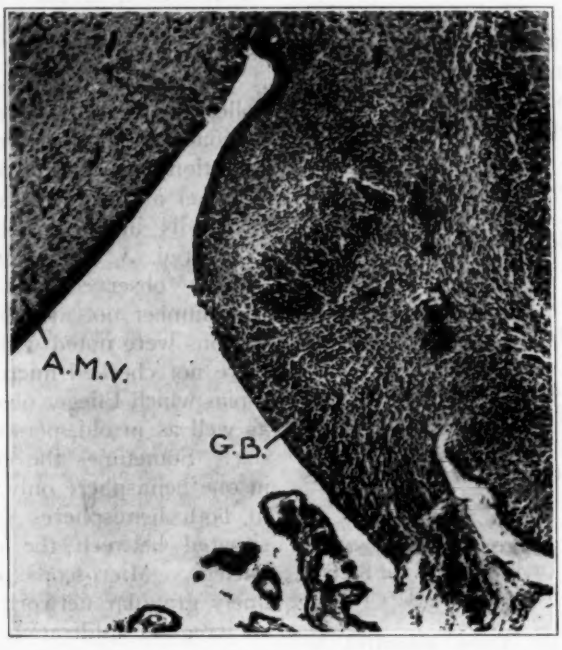


Fig. 2.—Normal appearance of the germinal bud in the cerebellum shown in figure 1. In this figure, and in the accompanying figures *A. M. V.* indicates the anterior medullary velum. *G. B.* designates the germinal bud. Hematoxylin and eosin stain; × 30.

Wohlwill [4] noted that a favorite site of rests was the roof of the fourth ventricle, and he com-

4. Wohlwill, F.: Zur Frage der sogenannten Encephalitis congenita (Virchow), Ztschr. f. d. ges. Neurol. u. Psychiat. **68**:384-415, 1921.

5. Wanke, R.: Zur Anatomie und Chirurgie des Medulloblastoms, Arch. f. klin. Chir. **177**:528-564, 1933.

6. Winkelman, N. W., and Eckel, J. L.: Midline Cerebellar Tumors ("Medulloblastoma"), Surg., Gynec. & Obst. **63**:372-381, 1936.

of embryonic development account for the elements of the germinal epithelium remaining in the roof of the fourth ventricle and that these elements may be ready to be activated under favorable and proper conditions. Ostertag [7] commented on abnormalities in the cerebellum and the possibility that tumors may develop from them.

OCCURRENCE OF CELL RESTS IN THE CEREBELLUM

The material examined in this study consisted of the cerebellums of 161 embryos, fetuses and infants; the youngest embryo was one of 8 weeks, and the oldest child was 5 years of age. It was obviously impractical to make serial sections of the entire cerebellum; therefore, we cannot state definitely that cell rests did not occur in regions of the cerebellum other than those in which we saw them. However, during the course of this study, hundreds of microscopic sections of the hemispheres and the vermis of the 161 cerebellums were examined, and the only region in which an abnormal collection of cells was seen was the vicinity of the posterior medullary velum. Sections from 104 of the 161 cerebellums included the region of the posterior medullary velum.

The germinal bud, from which the external granular layer is derived, may be seen in the region of the posterior medullary velum in sections of the fetal cerebellum (figs. 1 and 2). Sections were studied to determine the age at which the germinal bud normally disappears. Of the 104 cerebellums from which sections were cut showing the posterior medullary velum, 37 were from fetuses young enough to show the germinal bud. During the last part of intrauterine life this structure becomes much less definite. In no case was it observed after the infant had reached the age of 1 month.

Of the 104 cerebellums from which sections had been cut to show the region of the posterior medullary velum, 23 had abnormal collections of cells in that area. Five of these 23 cerebellums were obtained from fetuses of less than 9 months, and 6, from infants that were less than 1 month

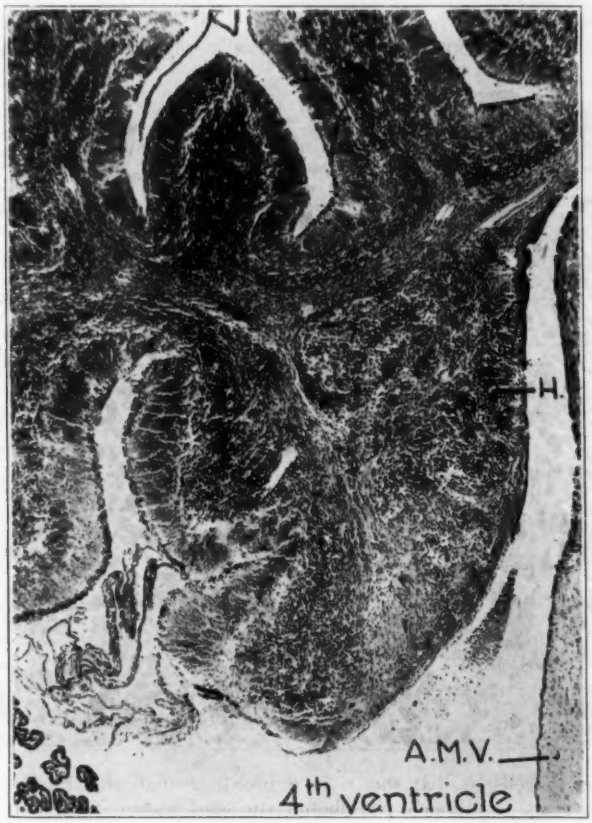


Fig. 3.—Section of the cerebellum of the fetus of 8.5 months, from the region of the posterior medullary velum. *H* indicates an abnormal collection of cells scattered helter-skelter. Mallory's phosphotungstic acid hematoxylin stain; × 33.

7. Ostertag, B.: Einteilung und Charakteristik der Hirngewächse: Ihre natürliche Klassifizierung zum Verständnis von Sitz, Augbreitung und Gewebsaufbau, Jena, Gustav Fischer, 1936.

of age. Since the germinal bud sometimes persists until the first month of postnatal life, the question arises whether the collections of cells in the region of the posterior medullary velum in these 11 brains were abnormal. We think these cells were abnormal because their morphologic appearance and arrangement were not those of a normal germinal bud. In 1 of these cerebellums the appearance was actually that of neoplasm. The cells simulated those seen in medulloblastoma, and there was a tendency to the formation of pseudorosettes. Many mitotic figures also were noted. Figures 3 and 4 show an abnormal collection of cells in a section of the cerebellum of a fetus of 8.5 months. In this section, various types of cells, including Purkinje cells, were scattered helter-skelter, and we were led to the conclusion that this mass of cells was not a germinal bud but an abnormal collection of cells. The presence of Purkinje cells in this region supports the opinion expressed by some authors that these cells arise not only from the primary germinal layer but from the external granular layer.

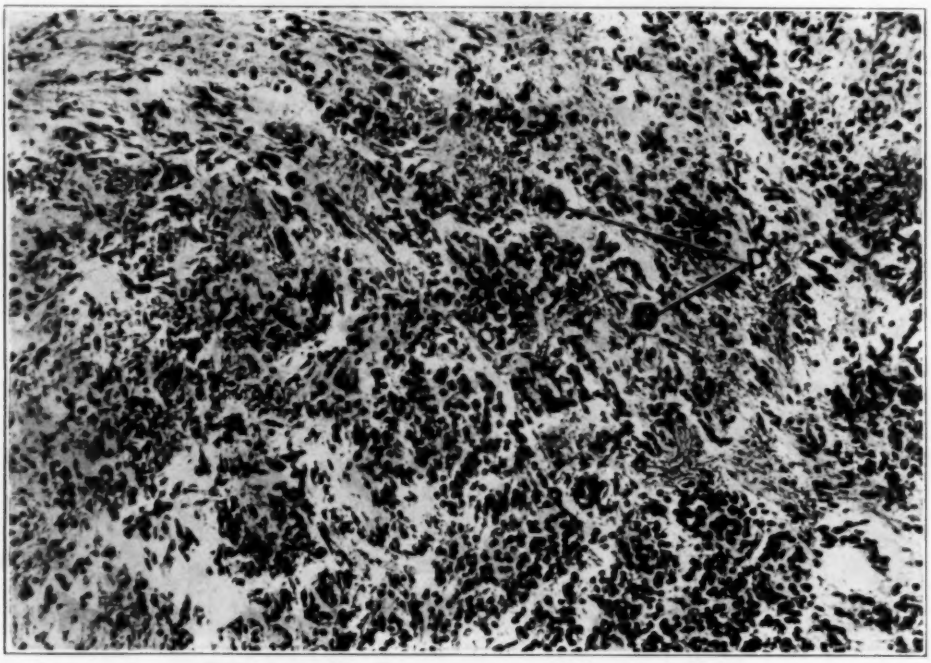


Fig. 4.—Same collection of cells as that shown in figure 1. *P* indicates Purkinje cells scattered among the other cells at irregular intervals. Mallory's phosphotungstic acid hematoxylin stain; × 190.

For the remaining 12 cerebellums in which abnormal collections of cells were seen, the age distribution of the infants was as follows: One infant was 1 month; 1, 2.5 months; 1 (fig. 5), 5.5 months; 1, 6 months; 2, 9 months; 1, 11 months; 2, 2 years; 1, 2.5 years; 1, 3 years, and 1, 4.5 years of age. It is evident, therefore, that 12 of the 23 infants or fetuses with abnormal collections of cells in the posterior medullary velum were past the age at which the germinal bud usually disappears.

That such a condition occurs even among adults is shown by the presence of an abnormal collection of cells in the posterior medullary velum of the cerebellum of a man aged 24 (fig. 6), who died ten days after an exploratory craniotomy for a pinealoma. There was no connection, grossly or microscopically, between the neoplasm and the cells in the posterior medullary velum.

The microscopic appearance of these abnormally placed cells varied with age. The immature appearance was more pronounced in young persons. In all cases the cells were scattered helter-skelter, and there was no suggestion of an orderly arrangement. At times Purkinje cells were present among the abnormally placed cells, and rosettes were observed occasionally.

SITE OF ORIGIN OF MEDULLOBLASTOMA

Bailey and Cushing [8] commented on the frequency with which a very cellular tumor occurs in the midline of the cerebellum. In 1924 they designated this tumor as "spongioblastoma cere-

8. Bailey, P., and Cushing, H.: Medulloblastoma Cerebelli: A Common Type of Midcerebellar Glioma of Childhood, Arch. Neurol. & Psychiat. **14**:192-224 (Aug.) 1925.

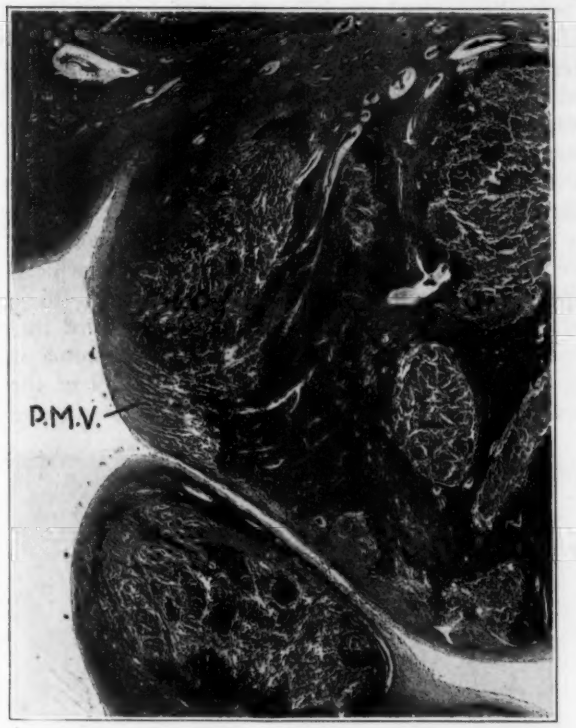


Fig. 5.—Abnormal collection of cells in the region of the posterior medullary velum (*P. M. V.*) in the cerebellum of an infant aged 5.5 months. Hematoxylin and eosin stain; × 15.

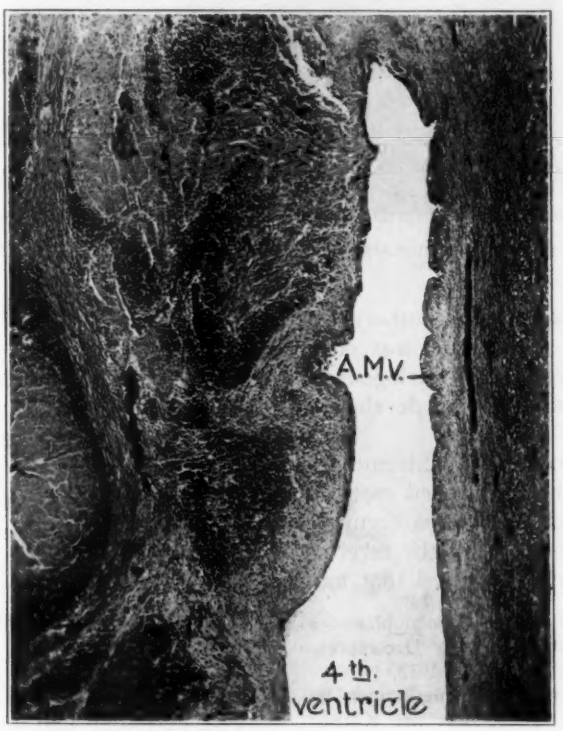


Fig. 6.—Abnormal collection of cells in the cerebellum of a man aged 24. Hematoxylin and eosin stain; × 45.

belli," since they were unaware that Strauss and Globus [9] had used the term "spongioblastoma" to indicate a cerebal tumor of a different type. Bailey and Cushing later decided to designate the tumor they described as "medulloblastoma," and they pointed out that in all the 29 cases which they reported the neoplasm originated from the roof of the fourth ventricle and that it developed as a central intracerebellar lesion. Macroscopically, the tumor was fairly well circumscribed, but microscopically it showed invasive properties. The cells were round or pear shaped, with scanty cytoplasm and oval nuclei; they were usually unipolar but sometimes bipolar. In some places they occurred in strands, in a palisade arrangement, or formed rosettes. There was a scant amount of connective tissue. The authors expressed the opinion that this tumor was composed mainly of "indifferent cells," analogous to those observed by Schaper [10] in the developing cerebellum.

Many authors have denied the existence of medulloblasts, and some have reported cases in which they thought a medulloblastoma occurred in a region of the brain other than the cerebellum. Kershman,[11] however, observed that medulloblasts do occur in the cerebellum but not in the cerebrum and the spinal cord. He expressed the opinion that this observation was in keeping with the fact that the medulloblastoma occurs exclusively in the cerebellum.

Since we encountered abnormal collections of cells in the region of the posterior medullary velum in 23 of 104 cerebellums which we examined in that area, we subsequently made a gross and microscopic study of the cerebellum in 25 cases in which the patient had died of medulloblastoma, to determine if possible the point of origin of the tumor. In all 25 cases the medulloblastoma was situated in the fourth ventricle. In 9 of the 25 cases the growth of the tumor was so extensive and the medulloblastoma had invaded the cerebellum to such an extent that it could not be definitely stated from what region it had originated. In the remaining 16 cases it could be stated that the growth had originated somewhere in the roof of the fourth ventricle, and in 8 of these 16 cases gross and microscopic examination suggested that the tumor had its origin in the posterior medullary velum (fig. 7). There was no evidence in the remaining 17 cases that the tumor might not also have arisen in the posterior medullary velum.

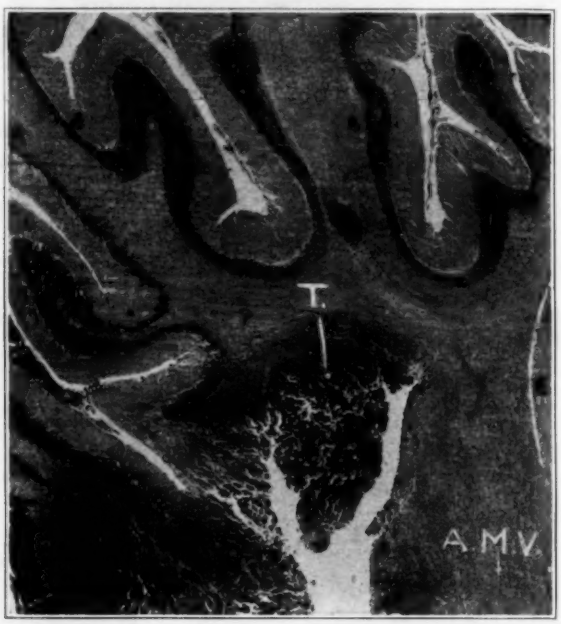


Fig. 7.—Medulloblastoma (*T*), which appears to have originated posterior to the fastigium. Hematoxylin and eosin stain; × 10.

9. Strauss, I., and Globus, J. H.: Spongioblastoma with Unusually Rapid Growth Following Decompression, Neurol. Bull., New York **1**:273-281, 1918.

10. Schaper, A.: Einige kritische Bemerkungen zu Lugaro's Aufsatz: Ueber die Histogenese der Körner der Kleinhirnrinde, Anat. Anz. **10**:422-426, 1895.

11. Kershman, J.: The Medulloblast and the Medulloblastoma: A Study of Human Embryos, Arch. Neurol. & Psychiat. **40**:937-967 (Nov.) 1938.

SUMMARY

The germinal bud from which the external granular layer of the cerebellum arises is situated at the posterior tip of the posterior medullary velum. It normally disappears before the end

of the first month of extrauterine life. In a study of the cerebellums of 161 fetuses and infants, abnormal collections of cells were observed only in the region of the posterior medullary velum. Of 104 cerebellums in which the posterior medullary velum was examined microscopically, 23 contained abnormal collections of cells. These cells were morphologically similar to cells of the medulloblastoma. In 8 of 25 cases of medulloblastoma the site of origin was the posterior medullary velum; in the other 17 cases the site of origin could not definitely be established, but it may also have been the posterior medullary velum. Since the site of abnormal collections of cells and the point of origin of the medulloblastomas were observed to be the region formerly occupied by the germinal bud, we think that the medulloblastoma arises frequently, if not exclusively, from cell rests which occur in the region originally occupied by the germinal bud.

833 S. W. Eleventh Avenue, Portland, Ore.
The Mayo Clinic, Rochester, Minn.

PHLEBOSTASIS AND PHLEBOTHROMBOSIS OF THE BRAIN IN THE NEWBORN AND IN EARLY CHILDHOOD

OTTO MARBURG, M.D., AND LOUIS CASAMAJOR, M.D
NEW YORK

GENERAL CONSIDERATIONS

In a discussion of injuries of the brain at birth three factors must be considered: the site of the lesion, its nature and its causes.

Site of Injury.—One must consider the site first, for, except for direct lacerations, all these injuries to the brain are localized in certain drainage areas of the venous system. Veins are more easily damaged than arteries, which are well protected where they enter the base of the skull. The base is already stabilized at birth, while the vault is still movable and easily injured.

The venous system in question includes various, somewhat independent, vessels. The regions involved in birth trauma are the drainage area of the great vein of Galen (vena cerebri magna) and that of the superior longitudinal sinus. The former vessel drains the blood from the centrum semiovale of the frontal and the anterior part of the parietal lobe via the anterior terminal vein, and from the posterior part of the parietal and the occipital lobe via the lateral ventricular vein. Even basal parts of the brain, the hippocampal region for instance, are drained by the great central vein (vena cerebri magna) via its basal branch (vena basalis of Rosenthal). Into the superior longitudinal sinus enter the cortical veins, which drain only the gray layer of the cortex and that part of the white substance which lies directly below the gray matter and consists of the U fibers and very small adjacent portions of the centrum semiovale.

One must take into consideration another factor concerning the entrance of the superficial veins into the sinuses, a factor which was particularly stressed by Cushing [1] as a source of hemorrhage in the newborn. The transition of the superficial veins into the sinus is indirect, via small bridging veins, which in earliest childhood are free, not connected with the cortex and therefore unprotected, but which in adults are provided some protection against injury by adhesions. Such a locus minoris resistentiae for the great central vein is its entrance into the sinus rectus. The sudden transition from a contractile to a rigid vessel, made almost at a right angle; the close relation to the tentorium cerebelli, with the possibility of easy obstruction in this region, and the undue tension on this part, particularly during delivery (Saenger [2]), explain the many lesions occurring in the area of the great central vein.

In summary, the following independent venous areas are the sites of predilection for lesions in the cerebral hemispheres.

1. The area of the great central vein (its branches, the vena terminalis anterior and the vena lateralis ventriculi), including the whole centrum ovale.

2. The area of the superior longitudinal sinus, i. e., the cortex with closely adjacent parts of the centrum semiovale.

3. Less significant for the lesions under discussion, the area of the sinus cavernosus and some ventral branches of the surface veins, i. e., the area ventral to the sylvian fissure.

Although the striopallidum and the optic thalamus are also drained, at least in part, by branches of the vena cerebri magna, these nuclei frequently remain undamaged in cases of injury to the great central vein. This may be evidence of a peculiar insusceptibility to damage of single branches of the veins (in this case, those of the vena cerebri magna) or of additional drainage by other vessels. Schlesinger [3] stressed a lack of ramifications in the drainage area of the terminal veins, which fact may explain the easier damage to the centrum semiovale. This inde-

Dr. T. J. Putnam gave advice in this study.

This work was aided by a grant from the Rockefeller Foundation.

From the Neurological Institute of New York; the Department of Neurology of the Columbia University College of Physicians and Surgeons, and the Research Laboratory for Neuropathology of Montefiore Hospital.

1. Cushing, H.: Concerning Surgical Intervention for Intracranial Hemorrhages of the Newborn, Am. J. M. Sc. **130**:563, 1905.

2. Saenger: Ueber die Entstehung der intrakraniellen Blutungen beim Neugeborenen, Arch. f. Gynäk. **120**:284, 1923.

3. Schlesinger, B.: The Venous Drainage of the Brain with Special Reference to the Galenic System, Brain **62**:274, 1939.

pendence of single branches is not uncommon; for example, single layers of the cerebellum may be affected independently via their venous supply (Pfeifer [4]). Whether there is a true independence of the various circulatroy areas, or whether the vasomotor systems of the various layers differ from each other, cannot yet be decided. Schwartz and associates [5] recognized all these factors as of great significance in the pathologic changes associated with injuries of the brain at birth. Thus, one may conclude that the site of birth injuries in the cerebral hemispheres is in certain areas of the venous drainage.

Pathologic Character.—An understanding of the site of injuries to the brain at birth makes the types of cerebral lesions easy to understand. There may, for instance, be hemorrhage or malacia due to a birth trauma which in its pathologic picture is scarcely distinguishable from that in the adult, or even the senile, brain. Possibly the lesser resistance of a not yet mature brain explains the larger defects, or the destruction may be somewhat more extensive, including even the glia and the mesodermal tissue (Spatz [6]). But usually one sees all the stages of resorption and repair, as in the adult brain (Marburg [7]). In addition to hemorrhage and malacia, a third pathologic change has frequently been under discussion, since Virchow,[8] in 1867, first described encephalitis congenitalis, the first portrayal of a nonsuppurative inflammatory process. At once the question arose whether the compound granule cells in the brain of the apparently normal newborn have a physiologic function, for building up the myelin sheaths (*Aufbauzellen* of Wohlwill [9]; Scheyer [10]), or a pathologic role (Schwartz [5b]; Neubürger,[11] and many others) or both (Wohlwill's *Aufbau- und Abbauzellen*). This question may be decided on the basis of the number and site of these cells. Infrequent cells along an axon may indicate a constructive, as well as a destructive, function, but destruction is followed by secondary degeneration, which is absent in constructive processes.

A destructive process is easy to identify by the focal arrangement of the compound granule cells, and the preservation of the ground structure of the tissue, while only the most vulnerable parts, the myelin sheaths and the axons, are destroyed. Occasionally the myelin sheaths alone are affected, with the well known picture of demyelination. Thus, all stages, from necrosis through malacia to degeneration of a whole fiber and to simple demyelination, are represented. In every case there is the same process of malacia in the area of a given venous drainage, but to different degrees. There is no doubt that in some cases the malacic process is accompanied by small hemorrhages, as well as by an exudation of lymphocytes and plasma cells into the walls of the blood vessels or the perivascular spaces. This commonly indicates an inflammatory process, and some authors have assumed that complications, such as infections (pneumonia) or septic processes, are the probable cause of the exudates. In other cases, however, there is exudation without such complications. Such an exudate can be explained only by an increased permeability of the blood vessel walls in the presence of protracted stasis, without too great a loss of the oxygen content of the blood (Binz [12]). The glia, which remains relatively intact, reacts by producing sclerosis, so that the destroyed nerve parenchyma is replaced. This diffuse sclerotic process resembles the well known diffuse sclerosis of the brain and is in site and structure similar to the condition in the cases first described by Heubner.[13] Its relation to

4. Pfeifer, R. A.: Grundlegende Untersuchungen für die Angioarchitektonik des menschlichen Gehirns, Berlin, Julius Springer, 1930.

5. (*a*) Schwartz, P.: Erkrankungen des Zentralnervensystems nach traumatischer Geburtsschädigung, Ztschr. f. d. ges. Neurol. u. Psychiat. **90**:263, 1942; (*b*) Die traumatischen Schädigungen des Zentralnervensystems durch die Geburt: Anatomische Untersuchungen, Ergebn. d. inn. Med. u. Kinderh. **31**:165, 1927. (*c*) Schwartz, P., and Berberich: Experimentelle Untersuchungen zur Frage des Geburtstraumas, Centralbl. f. allg. Path. u. path. Anat. **34**:621, 1923-1924. (*d*) Schwartz, P., and Fink, L.: Morphologische Entstehung der geburtstraumatischen Blutungen im Gehirn und Schaedel des Neugeborenen, Ztschr. f. Kinderh. **40**:427, 1926.

6. Spatz, H.: Ueber degenerative und reparatorische Vorgänge nach experimentellen Verletzungen des Rückenmarks, Ztschr. f. d. ges. Neurol. u. Psychiat. **58**: 327, 1920.

7. Marburg, O.: Die traumatischen Erkrankungen des Gehirns und Rückenmarks, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 11, p. 1.

8. Virchow, R.: Zur pathologischen Anatomie des Gehirns: I. Congenitale Encephalitis und Myelitis, Virchows Arch. f. path. Anat. **38**:129, 1867.

9. Wohlwill, F.: Zur Frage der Encephalitis congenita (Virchow), Ztschr. f. d. ges. Neurol. u. Psychiat. **73**:360, 1921.

10. Scheyer, H. E.: Ueber Fettkörnchenzellbefunde im Rückenmark von Föten und Säuglingen, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:185, 1925.

11. Neubürger, K.: Ueber die sogenannte Gliaverfettung im Grosshirnmark bei Kindern, Ztschr. f. d. ges. Neurol. u. Psychiat. **97**:598, 1925.

12. Binz, C.: Der Anteil des Sauerstoffs an der Eiterbildung, Virchows Arch. f. path. Anat. **59**:293, 1874.

13. Heubner, O.: Ueber diffuse Hirnsklerose, Charité-Ann. **22**:298, 1897.

what is called Schilder's [14] disease (encephalitis periaxialis diffusa) is obvious.

Etiologic Factors.—There may be many different causes of these injuries. Since certain venous systems are affected, with the usual lesions of the blood vessels, namely, hemorrhage and malacia, such injury may occur even in normal delivery. The excessive tension on the venous system, particularly at the junction of the great cerebral vein and the sinus rectus; compression by the tentorium, particularly during difficult labor, and protracted and instrumental birth may be other causes of the changes described. It is irrelevant whether the overburdening of the venous system alone, as suggested by Saenger,[2] or the difference in tensions inside and outside the uterus, which Schwartz [15] called *Minderdruckwirkung,* or both are factors in the production of the lesions. Asphyxia, obviously, plays a supporting, but never a decisive, role. The experiments of Thorner and Lewy [16] demonstrated that the effect of anoxia is a rather general damage to the walls of the blood vessels, as well as to ganglion cells. Warwick [17] stressed that, in addition to traumatic vascular lesions produced by molding of the head and the resulting congestion and stasis, with rupture of veins, disease conditions in intrauterine life (hemorrhagic disease of the newborn, syphilis, congenital heart disease and toxemia) must be considered. Most authors, however, have claimed that such intrauterine disease is of little significance.

Whereas all the aforementioned factors are in the realm of possibility, two characteristics distinguish birth lesions of the brain from other injuries: (1) the occasional onset of the signs and symptoms long after birth, and (2) the progressive course of the process after birth, particularly the malacia.

It may have been for these reasons that Winkelman and Moore [18] described obvious birth injuries (malacia) in an infant as "progressive degenerative encephalopathy." They compared this disease with "swayback" in lambs, in accordance with the description of Innes and Shearer: [19] The latter authors claimed that the disease in lambs is usually congenital but that the signs and symptoms occur only during the first weeks of life, at the latest six weeks after birth. There is ataxic gait with spasticity of the legs, particularly the hindlegs, and no fever and blindness may occur. The course is progressive, and the termination is generally fatal, with bronchopneumonia. The pathologic changes are characterized by destruction of the white matter, with compound granule cells but no evidence of inflammation; not only the myelin sheaths but the axis-cylinders are destroyed, and even the glia presents signs of degeneration.

The term demyelination for this process does not seem justified to us, since it is a malacia, with complete destruction of the affected parts. Innes and Shearer [19] compared the process to Schilder's disease,[14] obviously merely because of the site of the lesion, for there could be no other resemblance. Winkelman and Moore [18] accepted this suggestion in part and stated: "There are many clinical and pathologic similarities between swayback and Schilder's disease."

As indicated in the introduction, it is necessary to study birth injuries from a new point of view. Since we have observed 2 cases of our own and have found many others in the literature, this study seemed worth while, particularly since it may be possible to prevent injury in some cases.

REPORT OF CASES

CASE 1.[20]—*History and Course.*—S. B., a 9 month old boy, was delivered normally. From birth he was never active, did not eat well and took no interest in the surroundings. At the age of 4 months convulsions set in, with opisthotonos. When he was being turned, he held his head backward and his back arched. When the neck was bent, he cried. There was general stiffening of the body. The pupils reacted normally, and the ocular movements were normal.

Laboratory Tests.—Urine: Urinalysis showed an acid reaction, normal specific gravity, a trace of albumin and no sugar. The benzidine test gave a positive reaction for blood. Microscopic examination revealed 3 to 5 red blood cells and 12 to 18 pus cells per cubic millimeter, as well as profuse bacteria, yeast cells and ammonium urate crystals.

Blood: A blood count showed 65 per cent hemoglobin and 3,570,000 red cells and 11,350 white cells per cubic millimeter, with 1 per cent juvenile forms, 57 per cent segmented forms (58 per cent neutrophils) and 42 per cent lymphocytes.

Spinal Fluid: The Pandy reaction was negative; the sugar measured 80 mg. and the chlorides 742 mg. per

14. Schilder, P.: Zur Kenntnis der sogenannten diffusen Sklerose (Ueber Encephalitis periaxialis diffusa), Ztschr. f. d. ges. Neurol. u. Psychiat. **10**:1, 1912.

15. Schwartz (footnote 5 *a* and *b*).

16. Thorner, M. W., and Lewy, F. H.: The Effects of Repeated Anoxia on the Brain, J. A. M. A. **115**: 1595 (Nov. 9) 1940.

17. Warwick, M.: Cerebral Hemorrhage of the Newborn, Am. J. M. Sc. **158**:95, 1919.

18. Winkelman, N. W., and Moore, M. T.: Progressive Degenerative Encephalopathy: Occurrence in Infancy, with Antenatal Onset Simulating "Swayback" of Lambs; Report of a Case, Arch. Neurol. & Psychiat. **48**:54 (July) 1942.

19. Innes, J. R. M., and Shearer, C. D.: "Swayback": A Demyelinating Disease of Lambs with Affinities to Schilder's Encephalitis in Man, J. Comp. Path. & Therap. **53**:1, 1940.

20. Dr. P. Rezek put at our disposition the history, as well as the brain, in this case.

hundred cubic centimeters, and the Kline reaction was negative.

The temperature was always febrile or subfebrile, ranging from 99 to 102 F. Finally, the child died of pneumonia, at the age of 9 months.

Autopsy.—Brain: The cranial vault was of normal size, and the bones were of the usual texture. In separating the bones from the dura mater considerable force was necessary. The dura remained intact. In opening the dura anteriorly, almost a pint (500 cc.) of a clear, brownish yellow fluid escaped. The brain was surprisingly small, and the surface was wrinkled, particularly in its anterior part. The cortical substance was leathery and light yellowish brown. It was atrophic throughout except for small triangular areas just lateral to the median fissure on either side.

Other Organs: There was bilateral bronchopneumonia. The liver was enlarged, with a thin, tense capsule. It was firmer than usual and of a light color. The parenchyma cut with increased resistance. The architecture of the liver was obscure, and there was moderate degeneration of the parenchyma. The heart and the large vessels were normal.

Microscopic Examination of the Brain.—The left frontal lobe was cystic. In the right frontal lobe a part of the centrum semiovale was preserved, particularly in the superior frontal convolution. Transverse sections showed what might be called the skeleton of the frontal lobe, the convolutions being reduced to a thin shell and so greatly collapsed that the surface was wrinkled. Caudally, close to the genu of the callosal body (fig. 1), the left superior frontal convolution appeared normal. On the right side, not only the superior frontal convolution but the gyrus cinguli, as well as adjacent parts, was well preserved and presented an almost normal appearance. The cystic degeneration, on the left side, as well as on the right, involved the greater part of the hemispheres, the cavities of which were crossed by bridges of brain substance.

Caudally, the corpus callosum, as well as the septum pellucidum, was preserved, and so were the caudate and lenticular nuclei (fig. 2). In this region the superior frontal convolution, on the left side, as well as the gyrus cinguli, was almost intact; on the right side only the latter was preserved. The corona radiata on the left side was well stained in Weigert preparations; on the right side it was sclerotic. The cortex and subcortex were transformed into a chain of cysts, some of which were without contents and others filled with tissue rests.

In a section taken caudal to the preceding section (fig. 3), the region of the parietal lobe resembled that already described except that the convolutions near the cortical edge, as well as the hippocampal region (cornu ammonis), were preserved on both sides.

More caudally the cysts were not so numerous as in the more anterior sections, and the dorsal convolutions were almost microgyric. The dorsal portion of the anterior part of the occipital lobe was microgyric. Instead of cysts, there was a cribrous state, and toward the occipital pole, true sclerosis. Some fiber systems were preserved at all levels, but it would lead too far to describe them in detail.

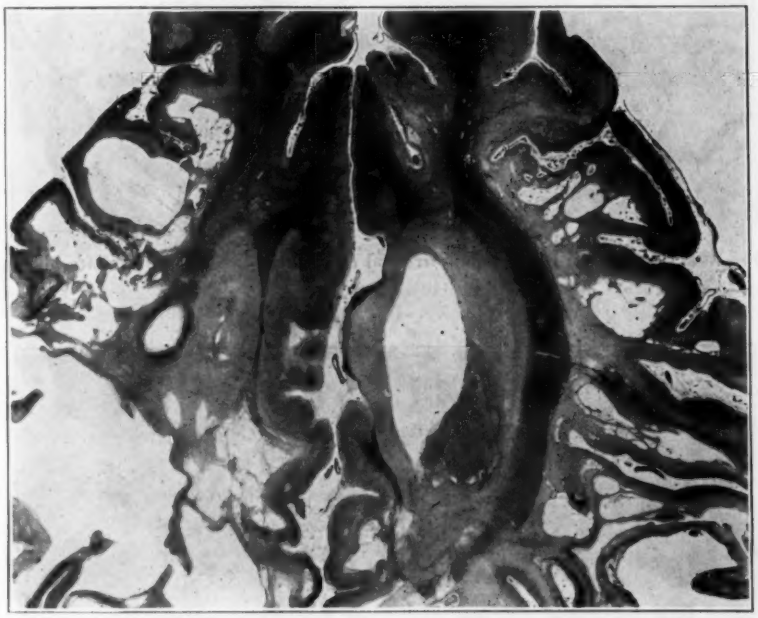


Fig. 1 (case 1).—Cystic degeneration of the centrum semiovale. Black spots left in the cortex indicate cortical thrombosis and softenings. Weigert stain.

In contrast to this destructive process within the hemispheres, the brain stem was intact except for secondary degenerations subsequent to the disintegration of the hemispheres. This was true of all parts, including the striopallidum (fig. 3) and, particularly, the cerebellum.

The areas of destruction corresponded to the drainage areas of the vena terminalis anterior and the vena lateralis ventriculi of the vena cerebri magna. The areas of the other tributaries of this vein, and in part the drainage area of the tributaries of the superior longitudinal sinus as well, were relatively intact.

In the destroyed areas foci, of irregular shape and size, were filled with necrotic debris. Another focus nearby consisted of compound granule cells with a

scarce network of tissue bridges between. A third area showed no vestige of fat granule cells, but only a loose network of glia fibers, resembling status cribrosus. Finally, there were oral or irregular foci of sclerosis. Although the plaques were not dense, they were recognized as sclerotic. The parenchyma in these foci, as well as the nerve fibers, myelin sheaths and axons, was completely destroyed. In addition to these evidences of a destructive process, there were cysts of various sizes and shapes. The walls of some of the cysts presented fat granule cells; other cysts were completely empty, and their walls were clean and even. Some cysts were filled to a minor degree with organized tissue rests, particularly blood vessels.

All these changes concerned the centrum semiovale. The cortical lesions were rather homogeneous in

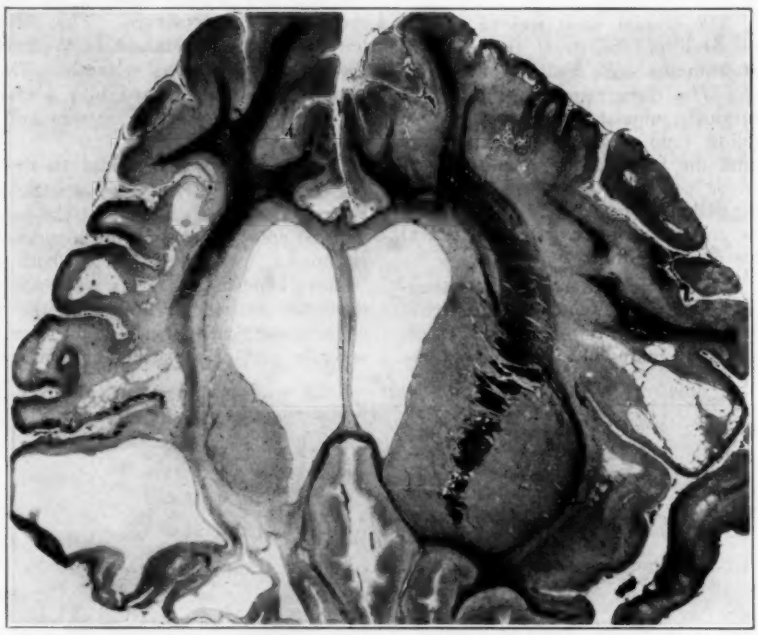


Fig. 2 (case 1).—Intact state of the superior frontal convolution and of the caudate nucleus and the putamen. Weigert stain.

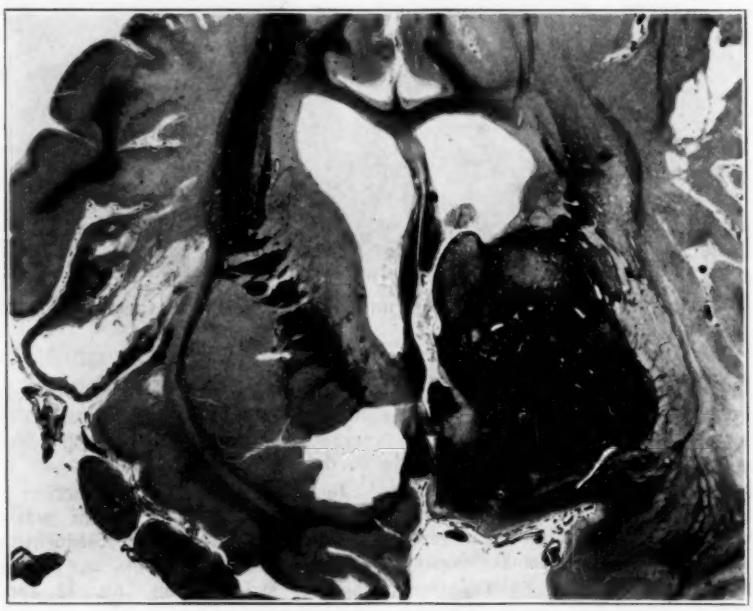


Fig. 3 (case 1).—Section caudal to that in figure 2, showing preservation of the optic thalamus, except for the dorsal lateral nucleus, and the striopallidum (right side), decrease in the softenings in the centrum semiovale (left side) and fresh malacias in the third frontal convolution on the left side. The black spots are areas of malacia. Weigert stain.

structure. There were only small foci in a chainlike arrangement, linked to each other by short bridges of typical areas of malacia. Some of these foci were accompanied by small, fresh hemorrhages, with a similar arrangement. In the frontal lobe, where the process had progressed farthest, the cortex was completely destroyed except for a thin seam represented by the molecular layer. At other levels, the gray matter of the cortex was preserved, and occasionally even the adjacent portion of the centrum semiovale. The continuous small foci resembled those in the well known état vermoulu (fig. 4).

The mesodermal tissue was also altered. There was slight fibrosis of the meninges, and the arteries in the meninges showed signs of endothelial growth (endarteriopathy) (fig. 4). Most striking were the many veins which, like straight wires, perforated the cortical substance to enter the meningeal veins (figs. 5 and 6). Some of these perforating veins were empty; some were filled with blood. Occasionally one could trace the course of such an empty vein as it emerged from a small focus (fig. 7). Sometimes thrombi occurred in these small veins (fig. 8). The small foci within the cortex occurred at different levels—some in the external portion of the gray matter and others below. The small veins in the region of the foci were empty. The arteries, on the contrary, particularly the arteries of the meninges, were congested. The degenerative process occurred at the roots of the small veins.

Other changes were of a mixed type. There were no signs of an inflammatory process and no exudative cells. Occasionally a fine pulverized sediment, perhaps calcium, lay near the focus; this stained dark blue in hemalum and eosin preparations. At some levels the

Fig. 4 (case 1).—État vermoulu in the cortex. The foci are in the second (resorptive) state. The vessels in the meninx show endarteritis. Hemalum-eosin stain.

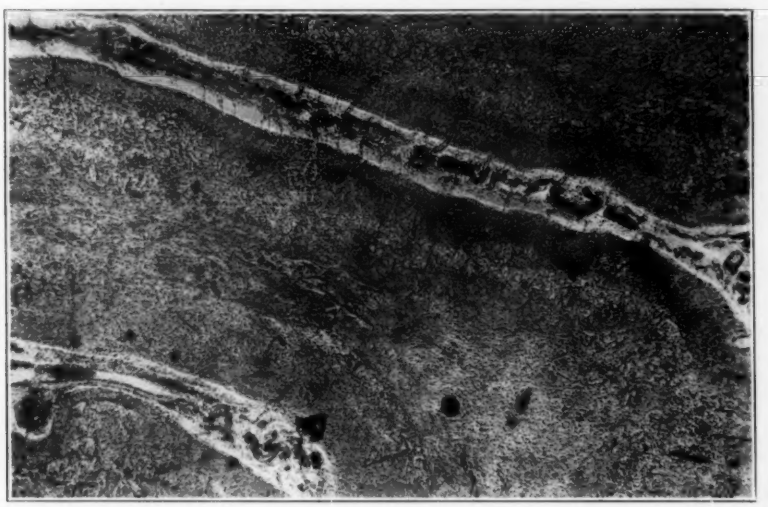


Fig. 5 (case 1).—Section of the cortex, showing empty veins spread toward the surface. Hemalum-eosin stain.

elastic membranes of the arteries were discolored, but it could not be decided whether there was true calcification.

In summary, there were areas of necrosis and malacia, partly acute, partly reparative, with final transition to sclerosis or cyst formation.

CASE 2.[21]—*History.*—L. W., a white boy, was delivered normally on March 23, 1933. Although breast fed, he did not gain weight. When he was 2½ months old, the parents noted that he did not move his left leg and that there was a slight curvature of the spine. The baby was irritable and fretful and whimpered whenever disturbed. He could not sit up or hold up his head.

In November 1934 he had pneumonia, and the manifestations increased. In April 1935 the child was admitted to the New York Neurological Institute. Examination revealed oxycephaly the circumference of the head being 36 cm., pronounced hypertonicity of all muscles, and severe spasticity, which was increased by any stimulus, such as touching of the skin or sudden noise. All the muscles were somewhat atrophic. The tendon reflexes were hyperactive on both sides. There was a Babinski sign bilaterally. Examination revealed advanced primary optic nerve atrophy, with a normal macula. The child showed no evidence of being aware of his surroundings. He fell asleep and awoke whimpering; when satisfied, he became calm. It is questionable whether he laughed. Frequently he coughed, shook, whimpered and cried, but quieted when his position was changed. The course of the disease was progressive. The slight opisthotonos observed at the first examination increased to a high degree toward the end of the illness (fig. 9). The same was true of

Fig. 6 (case 1).—Higher magnification of an area from the section shown in figure 4. Hemalum-eosin stain.

Fig. 7 (case 1).—Formation of foci in the cortex, showing compound granule cells and a round focus in the ventral portion, with an empty vein leaving its periphery.

21. Casamajor, L.: A Child Without a Forebrain, Tr. Am. Neurol. A. **63**:64, 1937.

the curvature of the spine and of the abnormal position of the limbs, namely, flexion of the arms with fist formation and extension of the legs with talipes equinovarus. Finally, there were complete stiffness, a tonic neck reflex and clonus of the hamstring muscles; all reflexes of the lower extremities were of grade 4. The child died of pneumonia, on May 2, 1936.

Autopsy.—The cerebral hemispheres were flabby sacks, the surface of which was shrunken like that of a dried pear. The pia mater was preserved over the entire surface; it was somewhat thicker than normal, and the blood vessels were fibrotic. The external configuration of the superior frontal convolution was preserved in its posterior half.

Microscopic Examination.—Transverse sections through the frontal lobe (original Weigert stain) showed complete destruction of the centrum semiovale. The ventral part was transformed into a single cyst; the dorsal part presented several smaller cysts, separated by tissue bridges.

More caudally the picture changed in that the superior frontal convolution was preserved (fig. 10). Some fibers were seen running from it into the internal capsule.

In the region of the knee of the corpus callosum, this body, as well as the septum pellucidum, was intact. The right hemisphere was cystic; the medial part of the left hemisphere and the caudate and lenticular nuclei on that side were preserved. On the right side these nuclei continued dorsally in brain tissue, which tapered toward the dorsal surface like the stalk of a plum.

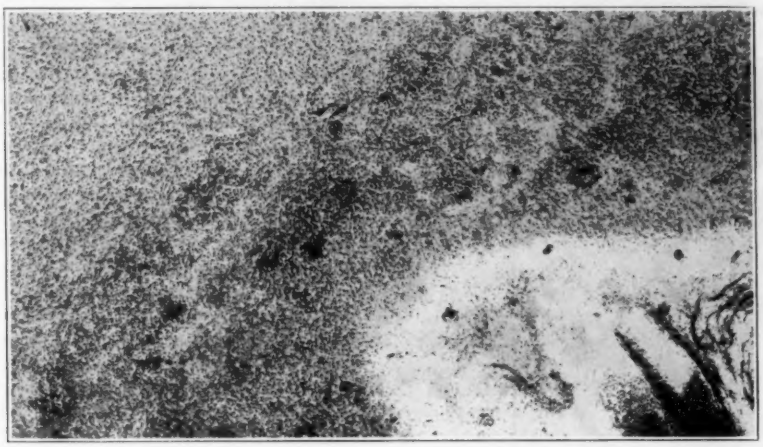


Fig. 8 (case 1).—Cortical foci. Thrombi appear as black spots in the veins.

Fig. 9 (case 2).—Final state of the process.

The head of the caudate nucleus in this section was sclerotic. The choroid plexus at this level was small, dense and atrophic, and its outer cells could not be distinguished.

The aforementioned changes were seen throughout both of the hemispheres. The wall of the sac was occasionally 1 mm., and sometimes 5 to 6 mm., in thickness. It consisted of a molecular layer and, where it was thickest, of cortical layers which appeared entirely normal. Occasionally only the upper layers of the gray matter of the cortex were preserved. The walls of the cysts were smooth and without evidence

of any reaction. In a few cysts only fat granule cells were noted in the wall. Other cysts contained homogeneous debris; in others, again, there was a slightly blue-stained (in hemalum and eosin preparations), homogeneous mass (fluid [?]). Occasionally a glial network spread from one wall to the other (fig. 11), and stumps of enlarged blood vessels stained blue with hemalum and eosin, the nearby choroid plexus being apparently shrunken; or empty blood vessels were present in large numbers. A few areas only resembled those in case 1, with a dense network of glia fibrils

Fig. 10 (case 2).—Transformation of the frontal lobe of the brain into an empty sac, the wall of which is of varying thickness. The superior frontal convolution is intact. Weigert stain.

(sclerosis, fig. 12) and no vestige of parenchyma. These patches developed in close proximity to cysts in which there were compound granule cells. The blood vessels were somewhat fibrotic but showed no changes in the intima. The vessels in the remnants of cortex were calcified and empty. The arteries were filled with blood; the veins were empty. In some places empty veins entered filled arteries. There were no thromboses.

In contrast to this almost complete destruction of the hemispheres, the brain stem was well preserved. The caudate nucleus and the optic thalamus on the right side were somewhat sclerotic, while the corresponding structures, as well as the lenticular nucleus, on the left side were intact. The optic chiasm was without myelin, so that the supraoptic commissures were distinct. The optic tract close to its entrance into the lateral geniculate body was well stained in Weigert preparations. Unfortunately, the part of the tract from the chiasm to this well stained portion was missing, so that it was impossible to state whether there was a focus in the tract, with preserved axons, the presence of which would explain the absence of secondary degenerations in this tract. From the midbrain caudally all parts were preserved except the cortical pathways to the respective regions (fig. 13). It may be stressed particularly that the vermis, as well as the lateral lobes, of the cerebellum were as well developed as those of a normal child. The region of the right dentate nucleus in some slides, presented brighter spots within the velum, but subsequent serial slides revealed that these areas were due merely to the staining method. Apart from the completely degenerated pyramidal tract, the medulla appeared normal except for the lateral half of the right olive and, to a lesser degree, the left olive (fig. 14). The hemalum and eosin preparations of this region (fig. 15) revealed pronounced contraction of the dorsal olivary vein before its entrance into the meninx. Behind this contraction the stem, as well as the branches, of the vein was enlarged and there was an area of disintegration in the olivary region corresponding to the demyelinated area. Thus, the process of phlebostasis was represented in its initial state.

The spinal cord was not available for examination.

In summary, the pathologic process in case 2 was similar to that in case 1, only far more advanced. In each case the process had the same site, with preservation of the same parts: the posterior part of the superior frontal convolution, the callosal body and the septum pellucidum. Also, the pathologic changes were similar—malacia, scleroses and cysts. The arteries were somewhat sclerotic. Part of the veins were empty, and part (particularly those emerging from the foci) were enlarged and filled with blood, with various stages of stasis.

The intensity and extension of the process differed in the 2 cases. In the first case not all branches of the vena cerebri magna were affected, as they were in the second case. There was evidence of a severe disturbance of the basal vein of Rosenthal, inasmuch as the optic nerve atrophy could not be explained otherwise.

COMMENT

As has already been mentioned, the site of a process is occasionally evidence of involvement of the venous system. In the 2 cases reported here the site corresponded to the drainage areas of the vena cerebri magna and the sinus longitudinalis superior. But whereas in the first case only the drainage system of the vena terminalis anterior and the vena lateralis ventriculi was damaged, in the second case the other branches of the great cerebral vein were also affected except for the vena septi pellucidi and some branches draining the corpus callosum and the posterior part of the superior frontal gyrus. This region was preserved in both cases. That other branches of the vena cerebri magna were

Fig. 11 (case 2).—Cystic degeneration. The cyst is filled with a fine network of glia fibers. In the dorsal portion are two enlarged veins; in the ventral portion a sclerotic choroid plexus appears.

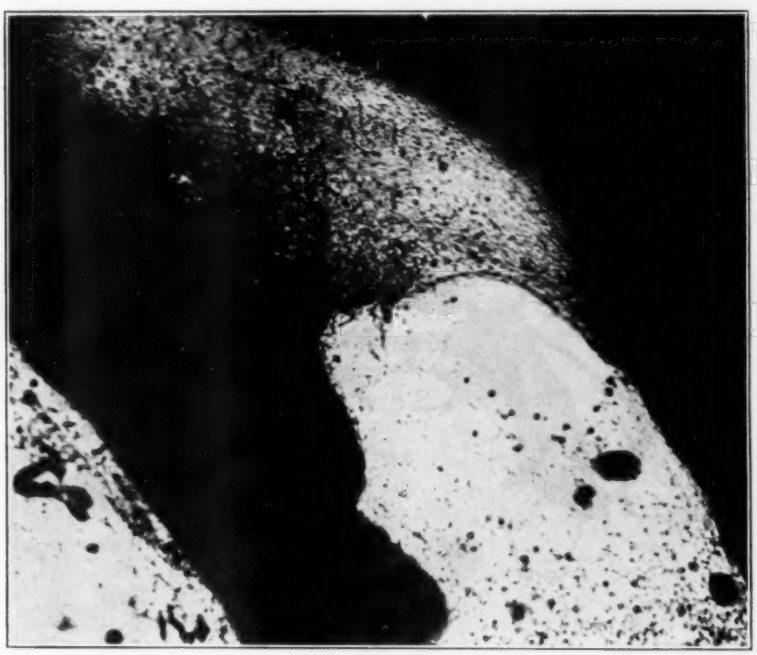


Fig. 12 (case 2).—Cyst, with scanty compound granule cells in the ventral part. In the dorsal portion are focus with effusion, an increase in glia and a beginning sclerotic plaque. Hemalum-eosin stain.

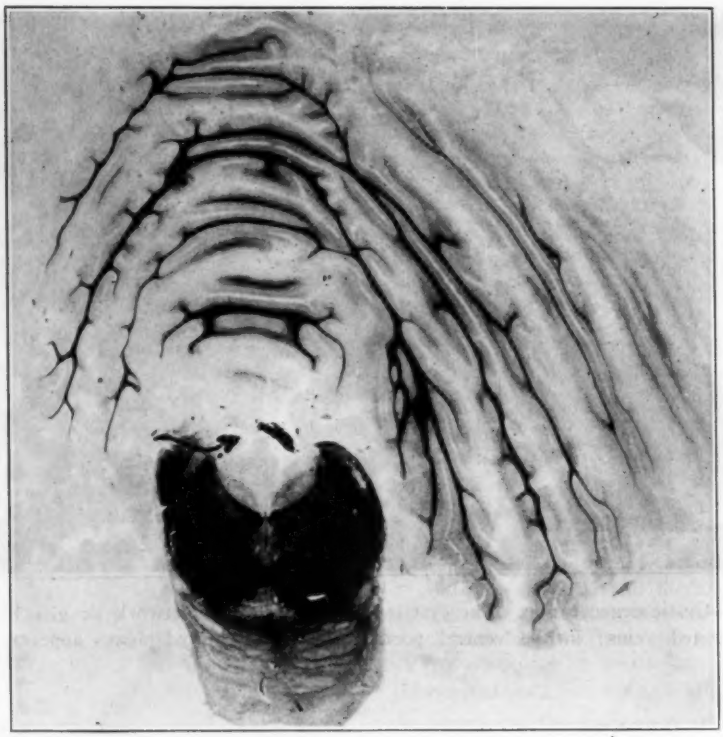


Fig. 13 (case 2).—Atrophy of the pons. All the afferent fibers are destroyed. The efferent fibers are pale. Weigert stain.

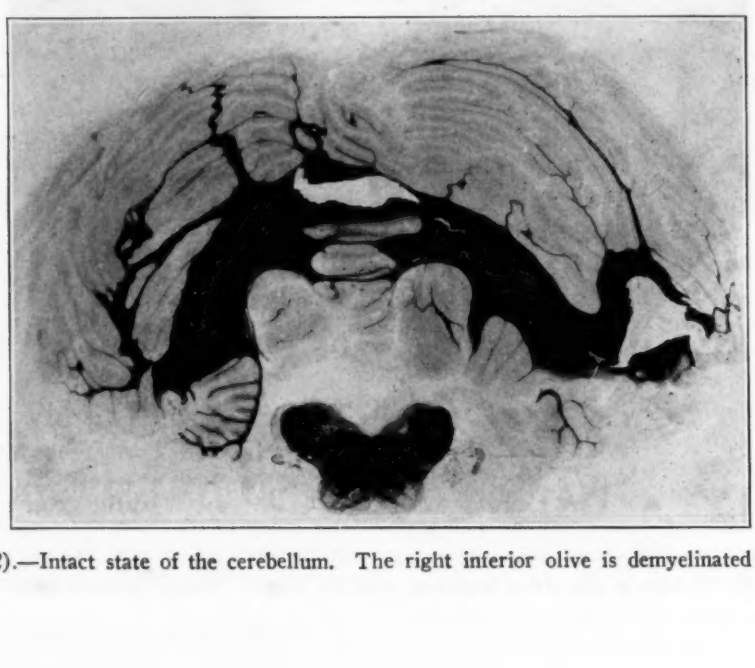


Fig. 14 (case 2).—Intact state of the cerebellum. The right inferior olive is demyelinated laterally. Weigert stain.

affected in the second case is evidenced by the fact that the hippocampal region had not been spared, in contrast to the condition in the first case; the optic nerve showed atrophic changes and the choroid plexus was sclerotic. These changes may be easily explained by the excessively long duration and the intensity of the process.

What was stated for the vena cerebri magna is true for the sinus longitudinalis superior. The destruction in the cortical area was far less intensive in the first case than in the second. But in both cases occasionally only the medullary layer was preserved; sometimes nearby the gray matter of the cortex was intact in its external portion, as well as the internal portion and the adjacent medullary area. Thus, in the cerebrum, as well as in the cerebellum, the venous plexuses which drain the molecular layer, the gray matter and the layer below it may be somewhat independent of each other, each plexus draining a separate area, or the vasomotor arrangements for these areas may differ.

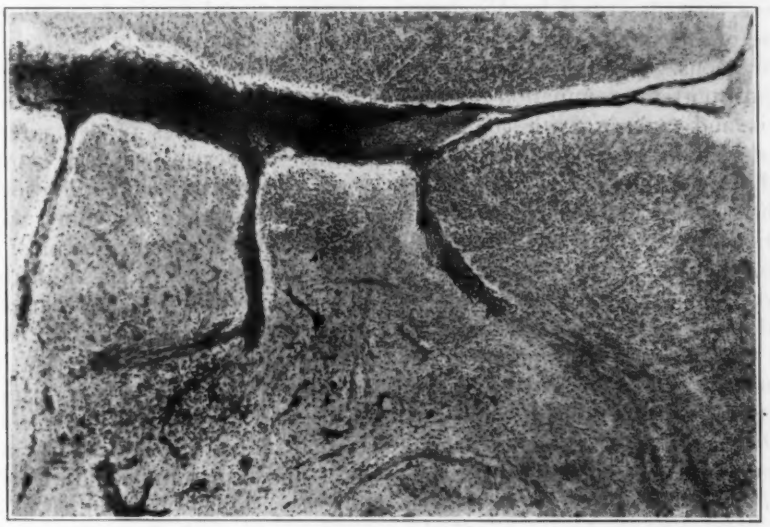


Fig. 15 (case 2).—Spasm of a vein, with retrograde stasis and degeneration of the right inferior olive. Hemalum-eosin stain.

The pathologic process coincided with a disturbance of the venous drainage. Typical areas of malacia led to formation of cysts, and incomplete repair resulted in cribrous glial scars (status spongiosus). In addition, malacic processes of minor intensity destroyed only the less resistant parenchyma, while the glia was preserved or occasionally formed a sclerotic focus.

That the process was restricted to damage in the drainage area of the venous system was proved by the many empty veins and venules leaving the foci of malacia, and particularly by the phlebostasis in case 2 (fig. 15), with constriction of the vein as its cause. The arteries were filled with blood, even when their walls were fibrotic or when a growth occurred in the endothelium of the intima. In case 1 thrombi were noted in some veins, but none were seen in case 2. Since in case 1 the pathologic process in the cortex was the younger, one may state two important facts: 1. The process resembled that known in senile brains as état vermoulu. 2. The process spread in the region of the venous plexuses, while the stems of the veins and the surrounding tissue remained free. According to figures 38 and 41 in Pfeifer's [4] monograph, there are three plexuses: The deep plexus drains the deep layers of the cortex and the adjacent medullary portion; the intermediate plexus, above the deep plexus, drains the superficial layers of the cortical gray matter, and the molecular layer is perforated by venules with a scanty plexus. It is self evident that these plexuses are not restricted to these respective layers but show transitions in distribution from one layer to the other, as indicated by the areas of malacias in case 1. It is certain that the process, at least in the cortex, begins in the venous plexuses described, in the roots of the small internal cortical veins.

These changes show a gradual transition from total necrosis to preservation of the more resistant parts, as the glial changes result either in cyst formation or in sclerotic plaques. The important question arises what kind of damage to the veins produces these changes. The answer would be simple if there were as many thromboses in the small veins as there are foci. But the large number of the foci is in contrast to the relatively

small number of thrombi, and it is almost impossible to state whether the thrombi are fresh or of long standing. Thus, one must concur with the statement of Ricker and Regendanz that the vasomotor system is affected, as evidenced in case 2 (fig. 15), and that stasis produces disturbances similar to those of thrombosis. One may conclude that phlebothrombosis or phlebostasis, or usually both, may produce the malacias mentioned, resulting in complete destruction of the nerve tissue, cyst formation, or incomplete destruction (parenchyma only), with formation of sclerotic plaques, in the drainage areas of the veins involved.

Before we discuss the causes and the pathogenesis of the lesions in our cases, similar cases in the literature may be mentioned. One of the first instances of this kind, that of lobar malacia of the hemisphere, was described by Virchow [8] (1867) as a case of encephalitis congenita. Delivery was spontaneous. The infant died when 6 days old, and postmortem examination revealed areas of necrosis and malacia, with compound granule cells, as well as lymphocytic exudates around the blood vessels—hence Virchow's designation, encephalitis. Cases of a similar, but more advanced, condition were described by Limbeck [22] (1886). In 1 case the infant lived only four days, and autopsy revealed cystic degeneration of the hemispheres and foci resembling those in Virchow's case. In a second case of Limbeck's, that of a 2½ year imbecile child with hydrocephalus, the disease seems also to have been congenital. Koeppen [23] (1898) described a case of degeneration of one occipital lobe, accompanied by a subdural hematoma, which he assumed to have been the cause of the damage to the brain. Similarly, Schmincke [24] (1919) suggested that in a similar case an obstruction of the lymphatics in the dura produced the necroses in the brain substance. Virchow's suggestion of an inflammatory process thus appeared unlikely, and a traumatic origin seemed more probable. Another case was described by Seitz.[25] A 29 year old woman, a cook, at the beginning of her third pregnancy, fell from the first floor into the yard. There was subsequent vaginal bleeding, but the patient recovered, without interruption of the pregnancy. In the fourth month of pregnancy she again fell, striking her abdomen. Again, bleeding set in, but the pregnancy was not disturbed; finally, after eleven hours of labor, an asphyxiated child was delivered spontaneously. The baby died in five hours, and postmortem examination revealed almost complete destruction of the hemispheres, which were changed into a flabby sac, filled with a clear, viscous, yellowish fluid. This case is evidence that injury to the abdomen of the mother during pregnancy may be followed by severe lesions of the fetal brain, which are limited almost exclusively to the hemispheres. In this case the process started in utero, as was possible in the cases of Virchow [8] and Limbeck.[22] Seitz concluded that there are two types of lobar disintegration, one of inflammatory and the other of traumatic origin.

Of note is Dahlmann's [26] case of cyst formation in the hemispheres of a 2 month infant (with normal delivery), which is similar to the case reported by Seitz, without, however, any previous trauma. Dahlmann explained the changes as the result either of encephalitis or of stasis, the latter being due perhaps to spasm of the arteries. In a case reported by Meier,[27] that of a 5 month old baby with total malacia of both hemispheres (obviously the same case as that reported by Hedinger [28]), two new factors appeared: the artificial birth (transverse presentation, external version and prolonged labor) and the onset of signs and symptoms eight weeks after birth. In this case, as in Dahlmann's [26] case, the diagnosis was apparently hydrocephalus. But in the case of Meier's the baby showed slight opisthotonos; hypertension, with flexion of the upper extremities, clenched fists and extension of the lower extremities, and talipes equinovarus. The first spinal puncture yielded a turbid fluid; the second, a bloody fluid. Meier described the pathologic changes as diffuse malacia with dilated blood vessels, without stating whether the arteries or the veins were dilated, and he referred to Dahlmann's case as a similar one.

22. Limbeck: Zur Kenntnis der Encephalitis congenita und ihrer Beziehung zur Porencephalie, Ztschr. f. Heilk. **7**:87, 1886.

23. Koeppen, M.: Ueber Gehirnkrankheiten der ersten Lebensperiode, als Beitrag zur Lehre vom Idiotismus, Arch. f. Psychiat. **30**:896, 1898; Ueber Veränderungen der Hirnrinde unter dem subduralen Hämatom, ibid. **33**:596, 1900.

24. Schmincke, A.: Ueber multiple Narbenbildung in der Grosshirnrinde kombiniert mit fibröser Leptomeningitis und Pachymeningitis haemorrhagica interna bei einem fünf Monate alten Kind, Ztschr. f. d. ges. Neurol. u. Psychiat. **51**:280, 1919.

25. Seitz, L.: Ueber die durch intrauterine Gehirnhämorrhagien entstandenen Gehirndefekte und die Encephalitis congenita, Arch. f. Gynäk. **83**:701, 1907.

26. Dahlmann, A.: Beitrag zur Kenntnis der symmetrischen Höhlen im Grosshirnmark des Säuglings, mit Bemerkungen über Entstehung von Hirnhöhlen im Allgemeinen, Ztschr. f. d. ges. Neurol. u. Psychiat. **3**: 223, 1910.

27. Meier, E.: Ueber einen Fall von totaler Erweichung beider Grosshirnhemisphären bei einen 5 Monate altem Kinde, Jahrb. f. Kinderh. **76**:552, 1912.

28. Hedinger, E.: Demonstration einer eigentümlichen Hirnerweichung bei einem fünf Monate alten Kinde, Centralbl. f. allg. Path. u. path. Anat. **23**:464, 1912.

A new avenue was opened by the investigations of Edinger and Fischer,[29] who stressed the value of physiologic study of children born without a forebrain. The anomaly in their case belongs to that under discussion here. A normally delivered child (after a labor of six hours) appeared normal at first, but four weeks after birth the parents observed that the movements were abnormal. As in other cases of this kind, opisthotonos and increased tonus developed. The pathologic process and its site were similar to those in the other cases, and the course was assumed to be a vascular process, beginning possibly in fetal life since the blood vessels showed round cell infiltration. Striking in this case were the duration of the disease for four years and the progressive course during that time.

Jakob [30] examined a 10 month old baby who was born asphyxiated after version from a transverse presentation. In this case the signs and symptoms, as well as the pathologic observations, were similar to those in our cases.

Hermel [31] termed the pathologic changes encephalomalacia chronica diffusa, a designation which is not quite justified, since he did not state whether the focus or the process was chronic. The focus is acute in cases of this disease, but the process occasionally lasts for years. Hermel's case was remarkable for the following reason: The onset of the disease was not immediately or a few weeks after birth, but occurred more than three years later. Thus, one cannot well blame instrumental delivery as the cause of the process, for the signs and symptoms developed in a manner similar to that in the other cases cited. The pathologic process did not present signs of inflammation, in spite of a slight increase in the temperature during the disease.

Wohlwill [9] discovered a new avenue of approach to the study of this process. He stressed trauma as the most prominent cause in his 5 cases, in 4 of which hemorrhagic pachymeningitis was present. But he further emphasized the importance of the veins since the process spread in their drainage areas. Obstruction (thrombosis) played a more minor role than phlebostasis, which explains not only the process itself but its progression. Wohlwill expressed agreement with Ricker's fundamental observations. Finally, he pointed to an intimate relation of the process in these cases to diffuse sclerosis and Schilder's disease.

The investigations of Schwartz [15] brought the solution of most of these questions. He claimed that injury at birth is a cause even in normal delivery, the difference between the pressure inside the uterus and that outside at the moment of presentation of the head (*Minderdruckwirkung*) being the chief factor. In almost every case a definite venous drainage area was affected—the area of the anterior terminal vein and the lateral ventricular vein of the great cerebral vein of Galen (centrum semiovale) and that of the veins entering the superior longitudinal sinus (cortex). Schwartz [5b] showed, in addition, the transition of the pathologic changes, from necrosis to malacia, with or without preservation of the more resistant parts (glia). Occasionally the process was so mild that only the myelin sheaths were destroyed, with resulting focal demyelination. The cases reported by Schwartz [5b] were of the same type as the cases of the other authors. He claimed with certainty that lobar sclerosis and diffuse sclerosis belong in the same pathologic group as diffuse malacia.

This is true of one of the best known cases of diffuse sclerosis, that of Heubner,[18] in which the process appeared after trauma to the head. It is also true of recent cases, those of Globus and Strauss [32] for instance, whose first case resembled that of Heubner,[18] the signs and symptoms developing also after repeated trauma. That does not mean that these authors claimed that a condition like that described is necessarily a sequela of a traumatic lesion. Some infections seem to have the same results with respect to the site and size of the damage. It has been generally stressed that syphilis does not play a role in production of this disease. But in a case reported by Schob [33] the mother had "florid syphilis" during pregnancy. The baby was delivered normally but died at the age of 2 months. There was typical complete cystic degeneration of the hemispheres, the result of necroses and malacias, and many compound granule cells, as well as plasma cells and lymphocytes, were noted around the blood vessels. There were no signs of syphilis in the nervous system, and the Wassermann reaction of the blood was negative. The slight inflammatory process had begun in the course of an intestinal disorder which the child had.

A case of Brocher's [34] belonging in this group was that of an 11 month old boy born prema-

29. Edinger, L., and Fischer, B.: Ein Mensch ohne Grosshirn, Arch. f. d. ges. Physiol. **152**:535, 1913.

30. Jakob, A.: Die extrapyramidalen Erkrankungen, Berlin, Julius Springer, 1923, p. 250.

31. Hermel, H.: Ueber einen Fall von Encephalomyelomalacia chronica diffusa bei einem vierjährigen Kinde, Deutsche Ztschr. f. Nervenh. **68**:335, 1921.

32. Globus, J. H., and Strauss, I.: Progressive Degenerative Subcortical Encephalopathy, Arch. Neurol. & Psychiat. **20**:1190 (Dec.) 1928.

33. Schob, F.: Totale Erweichung beider Grosshirnhemisphären bei einem zwei Monate alten Säugling, J. f. Psychol. u. Neurol. **40**:365, 1930.

34. Brocher, J. E. W.: Polyporencephalie, Ztschr. f. d. ges. Neurol. u. Psychiat. **142**:107, 1932.

turely, his twin sister being macerated. The clinical picture was that of hypertonia and cystic degeneration of the hemispheres. Brocher suggested that the cause was a venous circulatory disturbance resulting from trauma at birth, similar to that in the typical cases reported by other authors, particularly Schwartz.

Hallervorden,[35] in his report on the role of circulatory disturbances in the production of congenital idiocy, mentioned 3 cases of the kind under discussion—that of a 7 year old boy with complete malacia of the hemispheres; that of a 2¼ year old boy born prematurely, and, finally, that of a 6 year old boy with typical cystic degeneration of the hemispheres. The observation of 3 such cases in a relatively small series is evidence of the frequency of such damage.

The condition in cases such as those described by Mackay[36] is congenital and belongs to Schwartz's[5b] *Auflockerungsprozesse,* in which the ground structure of the tissue remains intact and only the most vulnerable parts are destroyed. The term "demyelinating encephalopathy," used by Mackay, is not justified, since, for example in the cases of Globus and Strauss,[32] the axons were also almost completely destroyed. The glia reaction in this condition obviously depends not only on the age but on the general state of the patient, since in young persons and in excessively marasmic states the glia does not react as usual.

A typical case of phlebothrombosis and phlebostasis in early childhood was described by Winkelman and Moore.[37] There had been prolonged labor, and resuscitation of the child was difficult. Three days after birth the child did not suck but could cry. Spinal puncture revealed a bloody fluid. Later the temperature rose to 104 F.; convulsions set in, and intensive rigidity and exaggerated tendon reflexes (bilateral Babinski sign), with loss of the swallowing reflex, were present. In twelve weeks the child died. Except for complicating meningitis, the anatomic picture was that of cystic necrosis of the hemispheres with intact brain stem, the affected areas corresponding to the drainage area of the vena terminalis anterior and the vena lateralis ventriculi. There was an enormous number of fat granule cells, as well as a beginning glial reaction with formation of sclerotic islands.

Involvement of parts of the cerebellum is unusual in cases of this condition. Calcified ganglion cells are occasionally seen in traumatic lesions. In spite of many clinical and pathologic similarities, Winkelman and Moore[18] did not liken the condition in their case to diffuse sclerosis or to Schilder's disease, as many other authors have done, particularly Wohlwill[9] and Schwartz,[15] but they pointed out its similarity to "swayback" of lambs, a disorder related to diffuse sclerosis which obviously occurs in man (Innes and Shearer[19]).

Our 2 cases also belong to the condition under discussion. Though apparently normally delivered, a few weeks after birth the children showed the first signs of a disease which terminated in an excessive increase in muscle tonus, opisthotonos, *arc du ciercle* position, flexed arms, stretched legs, clenched fists, talipes equinovarus, convulsions and exaggerated reflexes—in brief, the same picture as that in the other cases described. The areas of destruction in the brain coincided with the drainage areas of the aforementioned veins. The process was one of progressive malacia, followed by sclerosis or cyst formation, the final lesion depending on the amount of destruction of the tissue. Thus, this disease represents the pure form of encephalopathy due to phlebostasis and phlebothrombosis, no matter whether one calls it encephalomalacia chronica diffusa (Hermel[31]), polyporencephaly (Brocher[34]), congenital demyelinating encephalopathy (Mackay[36]), progressive degenerative encephalopathy (Winkelman and Moore[18]) or even "swayback."

By no means a complete citation of the literature has been made. Many similar cases have been described under the heading of cerebral palsy of early life (Sachs and Peterson,[38] case 7) or cerebral infantile paralysis. Sachs and Peterson stressed the role of birth trauma in such cases.

This brief review of the literature leads to the conclusion that trauma plays a major role in the cerebral disease described. But the trauma need not be a birth injury, as stressed by Wohlwill[9] and Schwartz[5]; it may occasionally occur during pregnancy, as evidenced by the case of Seitz,[25] or even after birth, as in the cases of Heubner[13] and Globus and Strauss.[32] An excellent example of trauma after birth was observed by Jaffé.[39] Immediately after delivery the baby was dropped to the floor from a height of about 30 inches

35. Hallervorden, J.: Kreislaufstörungen in der Aetiologie der angeborenen Schwachsinns, Ztschr. f. d. ges. Neurol. u. Psychiat. **167**:527, 1939.

36. Mackay, R. P.: Congenital Demyelinating Encephalopathy, Arch. Neurol. & Psychiat. **43**:111 (Jan.) 1940.

37. Dr. Winkelman sent us slides from his case (Winkelman and Moore[18]). Comparison of these sections with ours revealed complete identity.

38. Sachs, B., and Peterson, F.: A Study of Cerebral Palsies of Early Life Based upon an Analysis of One Hundred and Fifty Cases, J. Nerv. & Ment. Dis. **17**: 295, 1890.

39. Jaffé, R. H.: Traumatic Porencephaly, Arch. Path. **8**:787 (Nov.) 1929.

(75 cm.) and fell on her head. For hours the infant was unconscious and had convulsions, followed, after an interval of three months, by attacks of headache and fainting spells. Finally status epilepticus developed, with death from bronchopneumonia, at the age of 22 years. Postmortem examination of the brain revealed a cyst in the left occipital lobe, which was limited to the centrum semiovale, approximately in the drainage region of the vena lateralis ventriculi. Trauma at birth, however, is of the greatest importance when premature birth, instrumental delivery and even prolonged labor are considered. Unfortunately, many histories are inaccurate with regard to this point. In addition to trauma at birth, Schwartz,[15] as previously mentioned, stressed that normal delivery in itself, and even the beginning of presentation of the head, may suffice occasionally to produce the injuries described, as a result of *Minderdruckwirkung* and the thinned state of the vein of Galen and the superior longitudinal sinus. Such lesions occur in cases in which delivery is normal and labor is not prolonged—for example, in the case of Virchow,[8] in that of Dahlmann [26] and in case 2 in our report. Thus, the so-called normal birth does not necessarily mean that the brain is undamaged, and Sharpe and associates [40] observed a bloody cerebrospinal fluid in 13 of 100 normally delivered infants with no remarkable difference between the babies born of primiparas and those born of multiparas. According to Wohlwill,[9] the presence of a subdural hematoma, or even of pachymeningitis hemorrhagica interna, is evidence of a previous injury, as seen in 4 of his 5 cases and in the cases of Koeppen [23] and Schmincke.[24]

Another characteristic change may be added here—the resemblance of the cortical involvement in our case 1 to what in senile brains is called état vermoulu, and which is, according to Marie,[41] the sequela of severe arteriosclerosis. Spatz [42] attempted to prove its traumatic origin, and Scholz [43] indeed described similar changes after trauma in childhood. He noted that the depressions between the convolutions are affected in particular (see figure 12 in his paper).

Finally, the endarteriopathy in case 1 of the present paper and the arteriofibrosis in case 2 should be mentioned. Occasionally there is in lesions of traumatic origin an increase of the intima, called vasopathia by Marburg,[7] which was later described by other authors.

In spite of the prevailing incidence of trauma in the histories, one must admit that in some cases there is little evidence of any trauma as a cause of the cerebral damage. In the case of Hermel [31] the onset of the disease was a few months before the death of the 4 year old child. There had been a difficult delivery, with breach presentation, but for three and a half years the child was apparently normal. Thus, the time factor must be considered. In some cases the changes are so far advanced that the time after birth is too short to explain the changes on a basis of injury incurred at birth. That was true in Limbeck's [22] first case, in which the infant lived only four days and a cystic degeneration, similar to that in Seitz's [25] case, was present. On the contrary, in Virchow's [8] case the child lived five days, but autopsy revealed merely necrosis of the cerebral hemispheres. In the cases of Dahlmann [26] and Schob [33] it is difficult to decide whether the cystic degeneration occurred after birth or during intrauterine life. The clinical manifestations in the newborn are not decisive in this respect, for they develop occasionally rather late, coinciding with the late development of the respective parts of the brain. It may be six or eight weeks before the first signs and symptoms occur, as was first pointed out by Soltmann [44] (1876). Thus it is not surprising that in case 2 of the present study the signs did not begin to appear until eight weeks after birth.

In spite of all these factors, one must admit that trauma plays a predominant role in producing the destruction of the brain under discussion. The trauma may occur at birth, or the injury may be incurred before or after birth.

Virchow [8] called the disease inflammatory because he saw real exudates, as well as compound granule cells. There was no evidence of syphilis, as there was in the case of Schob,[33] who noted, in addition to compound granule cells, plasma cells and some lymphocytes in the perivascular spaces. But the role of syphilis as a cause of the cerebral disease in Schob's case is questionable, since the mother had a fresh infection and the Wassermann reaction of the baby's blood was negative. In

40. Sharpe, W., and Maclaire, A. S.: Intracranial Hemorrhage in the Newborn, Surg., Gynec. & Obst. **38**:200, 1924. Sharpe, W.: Intracranial Hemorrhage in the Newborn, J. A. M. A. **81**:620 (Aug. 25) 1923.

41. Marie, P.: Des foyers lacunaires de désintégration et des differents autres états cavitaires du cerveau, Rev. de méd. **21**:281, 1901.

42. Spatz, H.: Kann man alte Rindendefekte traumatischer und arteriosklerotischer Genese von einander unterscheiden? Die Bedeutung des "etat vermoulu," Arch. f. Psychiat. **90**:885, 1930.

43. Scholz, W.: Histologische Untersuchungen über Form, Dynamik und pathologisch-anatomische Auswirkung funktioneller Durchblutungsstörungen des Hirngewebes, Ztschr. f. d. ges. Neurol. u. Psychiat. **167**:424, 1939.

44. Soltmann, O.: Experimentelle Studien über die Funktionen des Grosshirns des Neugeborenen, Jahrb. f. Kinderh. **9**:106, 1876.

other cases complications or infection induced by spinal or cerebral puncture led to inflammatory reactions. Finally, one should not forget that occasionally white blood cells penetrate the walls of blood vessels by diapedesis, particularly the walls of the veins in the presence of prestasis. In any case one cannot exclude the possibility that, in addition to a traumatic form of hemispherical and cortical destruction, there are other forms, perhaps those of inflammatory origin, as suggested by Seitz [25] and Dahlmann.[26] In the present cases there was no evidence of inflammation, but neither was there any history of trauma. The pathologic picture in the present cases was, however, the same as that in the cases in the literature—first, areas of necrosis and malacia throughout the centrum semiovale, with complete, or almost complete, destruction of the entire tissue. This resulted in the formation of cysts or in a status spongiosus, with cystlike compartments. Side by side were fresh foci filled with compound granule cells; foci with almost completely resorbed cells, and foci free of cells, with even and clean walls and occasionally with fibrous tissue rests extending deep into the cyst. The glial reaction in these areas of necrosis and malacia was scanty. A second process consisted of small areas of sclerosis, which, though not dense, were certainly due to an increase of glia. The myelin sheath, as well as the axons, had disappeared. Some authors, however, in describing similar plaques, have claimed that the axons were preserved in these foci. Schmincke [24] suggested that the obstruction of the veins in the dura is followed by a disturbance of the circulation of the lymph, whereas Hallervorden [35] rightly stressed that in cases of prestasis there is a *seröse Durchtränkung* of the tissue, a serous exudation which destroys the less resistant parts of the tissue—the myelin sheaths and usually the axons, which may, however, sometimes be spared. That explains sufficiently the differences in the pathologic processes, though they are due to the same factor, namely, phlebostasis and phlebothrombosis, as proved in the 2 cases in this report. The same is true of the cortical destruction, resembling état vermoulu. Scholz [43] proved experimentally that passing spasm of a blood vessel followed by transient phlebostasis may produce the lesions described.

In addition to the similarities in site and pathologic changes, there is the similarity in the clinical pictures. Although the clinical descriptions in particular in these cases lack accuracy, similar significant signs and symptoms are recognizable. Slow movements of the limbs followed by paresis and paralysis, with exaggeration of the tendon reflexes and a Babinski sign, are observed in almost every case. In addition, there is usually an increase in muscle tonus, leading in cases of longer duration to complete stiffness and a characteristic posture, as described in case 2. These signs are frequently accompanied by convulsions, the type of which cannot always be recognized. In some cases the seizures are of clonic-tonic type, as in epilepsy. There may also be signs, though only occasionally, which may easily lead to a wrong diagnosis. Initially, there is often a peculiar weakness of the muscles of the neck, so that the head cannot be kept in an upright position but drops on the chest. Dollinger [45] called this phenomenon motor infantilism, or static infantilism. We assume that there is merely a disturbance in the tonic neck reflexes. A second phenomenon is the startling response of the flexors to any sensory or acoustic irritation—obviously a defense reaction occurring in cases in which the brain stem is preserved. Finally, there is sometimes optic nerve atrophy, as in case 2 of the present study. The cause of this disturbance could not be determined, since no specimens of the complete optic apparatus were available.

It is difficult to explain the course of the disease, which in the cases of long duration is progressive. In cases in which the trauma is a direct laceration or an arterial hemorrhage, the injury is usually followed immediately by signs and symptoms from the affected parts; but in cases with slowly progressive venous obstruction there are occasionally a long period of latency and slow progression of the signs and symptoms. The long period of latency may be explained in part by the particular reaction of the brain of the newborn, but possibly, in addition to the so-called late apoplexy associated with trauma, there is delayed malacia. One may assume that the vasomotor apparatus reacts late and that, once damaged, it remains irritable, so much so that any disequilibrium in the venous system causes a progression of the process. Case 2, particularly the contraction of the olivary vein, seems to prove this.

Through the investigations of Natus [46] and, particularly of Ricker and Regendanz,[47] as well as of Franklin,[48] an understanding of vasostasis, its causes and sequelae, has been acquired. Natus [46] observed that dilatation due to irritation

45. Dollinger, A.: Geburststrauma und Zentralnervensystem, Ergebn. d. inn. Med. u. Kinderh. **31**:373, 1927.

46. Natus, M.: Beiträge zur Lehre von der Stase nach Versuchen am Pankreas des lebenden Kaninchens, Virchows Arch. f. path. Anat. **199**:1, 1940.

47. Ricker, G., and Regendanz, P.: Beiträge zur Kenntnis der örtlichen Kreislaufstörung, Virchows Arch. f. path. Anat. **231**:1, 1921.

48. Franklin, K. J.: A Monograph on Veins, Springfield, Ill., Charles C Thomas, Publisher, 1937.

occurs earlier in the veins than in the arteries, and that constriction appears later, as in the capillaries. The small veins especially constrict earlier than the large and medium-sized ones. Extravasation occurs earlier from the veins than from the arteries. These statements of Natus, which on the whole were confirmed by Ricker and Regendanz,[47] explain the reaction of the veins in cases of trauma, as well as of infection; in addition, the particular disposition of the veins in the area of the vena cerebri magna, as well as that of the sinus longitudinalis superior, must be considered. This view has been generally accepted, though it has been proved by Joannovics [49] that another possibility exists. He demonstrated that the injection of brain emulsion into an animal with a traumatic lesion of the brain increases the softening process. In any case the brain of the newborn, more than the mature brain, is exposed to destructive processes from various sources. The site, the pathologic changes and the clinical picture obviously resemble those of so-called diffuse sclerosis. The latter term covers various morbid entities, among which the early infantile form is of particular interest with regard to the condition under discussion. For in cases of the infantile type the aforementioned triad of signs is extraordinarily pronounced. But even the cases occurring in early childhood present pathogenic differences, and only the cases with a well defined progressive degenerative process are to be considered here. The cases are sometimes sporadic, such as the one described by Globus and Strauss,[32] and sometimes familial, such as the cases described by Krabbe [50] and Eiselsberg.[51] It may be noted that case 3 of Globus and Strauss [32] resembles exactly in its clinical picture case 2 of the present study. Anatomically, the degenerative process is far more pronounced than the reconstructive process, since the glial increase in this case is least developed. We succeeded in finding the record of a sister of this patient in the collection of Dr. Charles Davison. She had the same disease as her brother, in a far advanced state, with thromboses of almost all the sinuses. The cases of Jakob and Scaravelli [52] (8 brothers and sisters with idiocy, deaf-mutism and quadriplegia spasmodica) do not belong to the type under discussion, whereas the 4 cases in a family described by de Lange [53] approach this type, in spite of the different opinions expressed by the latter author. The scanty number of compound granule cells she noted is possibly due to the lack of osmium-stained preparations. The glia cells she described did not differ in size and shape from glia cells in degenerative or reparative processes to such an extent that one must assume a blastomatous process. While this case is certainly exceptional, the other cases in the literature, particularly case 3 of Globus and Strauss, show a close relation to those under discussion.

We have little doubt that in some cases there is a close relation of diffuse sclerosis to the encephalopathy produced by phlebostasis and phlebothrombosis of traumatic origin, and possibly, likewise, to swayback in lambs. This close relation becomes obvious when one considers cases of diffuse sclerosis in which, in addition to the foci in the centrum semiovale, there are also lesions in the brain stem. The genesis of such foci in the brain stem is best illustrated by the lesion in the region of the inferior olive in case 2: phlebostasis due to obstruction of the wall of the vessel by constriction. According to Schlesinger,[3] tributaries of the vena cerebri magna drain parts of the cerebellum and the pons, which may explain the cerebellar involvement in the case of Winkelman and Moore.[18]

The cases just mentioned also form a link in the pathogenesis of multiple sclerosis suggested by Putnam and his co-workers.[54] As is well known, Putnam not only observed the production of foci by phlebothrombosis in cases of multiple sclerosis but experimentally produced such foci by various coagulants (Hoefer and Putnam [54c]). Thus, there is little doubt that obstruction of the veins may be the cause of diffuse and of multiple sclerosis, as it is the cause of the devastating processes observed in early childhood as sequelae of birth lesions and other injuries, and of various clinical manifestations.

This process of venous obstruction not only may be a disease entity but may occur sympto-

49. Joannovics, G.: Zur Wirkung fermentativ gewonnener Spaltungsprodukte aus Geweben und Bakterien, Wien. klin. Wchnschr. **33**:649, 1920.

50. Krabbe, K. A.: New Familial Infantile Form of Diffuse Brain Sclerosis, Brain **39**:74, 1916.

51. Eiselsberg, F.: Ueber frühinfantile familiäre diffuse Hirnsklerose, Ztschr. f. Kinderh. **58**:702, 1937.

52. Jakob, C., and Scaravelli, A.: A propósito de un caso de ocho hermanos con idiocia, sordomudez y cuadriplejía espasmódica familiar,. Rev. neurol. de Buenos Aires **5**:283, 1940.

53. de Lange, C.: Ueber die familiäre infantile Form der diffusen Gehirnsklerose (Krabbe), Ann. pædiat. **154**:140, 1940.

54. (*a*) Putnam, T. J.: Studies in Multiple Sclerosis: "Encephalitis" and Sclerotic Plaques Produced by Venular Obstruction, Arch. Neurol. & Psychiat. **33**: 929 (May) 1935. (*b*) Putnam, T. J.; McKenna, J. B., and Morrison, L. R.: Studies in Multiple Sclerosis: I. Histogenesis of Experimental Sclerotic Plaques and Their Relation to Multiple Sclerosis, J. A. M. A. **97**: 1591 (Nov. 28) 1931. (*c*) Hoefer, P. F. A.; Putnam, T. J., and Gray, M. G.: Experimental "Encephalitis" Produced by Intravenous Injection of Various Coagulants, Arch. Neurol. & Psychiat. **39**:799 (April) 1938.

matically. As Marburg [55] stressed as early as 1906, the foci of demyelination of dementia paralytica completely resemble those of multiple sclerosis, and Merritt, Putnam and Campbell [56] also noted venous obstruction in cases of the former disease. The foci in Binswanger's disease (progressive subcortical encephalopathy), resembling those in diffuse sclerosis, are obviously caused by a disturbance of the venous drainage, and not by any arterial trouble.

In addition to this close relation in some cases between diffuse sclerosis and cerebral lesions produced by traumatic phlebostasis and phlebothrombosis, there is a similarity of the latter condition with "swayback" in lambs. As previously mentioned, Innes and Shearer [19] noted the resemblance of "swayback" to diffuse sclerosis. But "swayback" resembles even more the process of phlebothrombosis and phlebostasis in the brain. For here, also, there is an absence of hereditary transmission; there is no difference between lambs born of a primipara and those born of a multipara; only one or two animals in a flock are diseased, the others being normal; the onset of the signs and symptoms occasionally takes place a long time after birth (up to eight weeks); there are differences in the intensity of the process, which in some cases leads to death and in others to healing with defects, and, above all, the pathologic processes are the same. All of which is evidence of the identity of "swayback" in lambs with the effects of phlebostasis and phlebothrombosis in the human brain. It is probable that some of the cases of "swayback" are caused by trauma, before, during or even after the delivery. But it cannot be excluded that this peculiar process may also have other, as yet unrevealed, causes.

Cushing [1] tried to cure hemorrhage occurring at birth by operation, with success in 2 of 4 cases. Operation is not possible in cases with necrosis, malacia or serous perfusion. What is necessary, therefore, is the prevention of injuries. Thorough care of the mother is essential from the beginning of pregnancy. The danger to the newborn is particularly great when there are several previous children, and voluntary helpers should be available in such homes. Delivery should be done carefully, preferably in an adequate hospital, where proper care for the newborn is easily available. Of course, injuries in early childhood cannot be avoided and will frequently occur, but they are not always followed by lesions of the brain. Yet many cases of later epilepsy may be traced to an injury incurred during childhood. The period of danger is during pregnancy, delivery and early childhood.

55. Marburg, O.: Die sogenannte akute multiple Sklerose (Encephalomyelitis periaxialis scleroticans), Leipzig, Franz Deuticke, 1906.

56. Merritt, H. H.; Putnam, T. J., and Campbell, A. C. P.: Pathogenesis of the Cortical Atrophy Observed in Dementia Paralytica, Arch. Neurol. & Psychiat. **37**:75 (Jan.) 1937.

CONCLUSIONS

Phlebothrombosis and phlebostasis are frequently the cause of severe disintegration of the brain in early childhood. Lesions of the veins and venous sinuses are predominantly caused by trauma before, during or after birth. But other causes, such as infection (?), cannot be excluded. The site of the lesions corresponds to the drainage areas of the veins: the centrum semiovale, the area of the vena terminalis anterior and the vena lateralis ventriculi of the great vein of Galen (vena cerebri magna); the cortex and adjacent parts of the meditullium, drained by the sinus longitudinalis superior.

The pathologic changes are necrosis, malacia and serous perfusion, followed by total or partial destruction of the tissue, with sparing of the most resistant parts (glia) and destruction of the least resistant structures (myelin sheaths and axons).

The repair depends on the degree of destruction: Complete destruction is followed by cyst formation, and incomplete destruction, by sclerosis.

Thus, the condition under discussion is closely related to diffuse sclerosis occurring in childhood, which to some extent is only the terminal stage of a phlebothrombotic or a phlebostatic state.

The same relation seems to apply to a disease in lambs called "swayback."

In prevention of this disturbance, particular care during pregnancy and delivery is necessary.

630 West One Hundred and Sixty-Eighth Street.

EXPERIMENTAL PRODUCTION OF FOCAL EPILEPSY

B. L. PACELLA, M.D.; N. KOPELOFF, PH.D.; S. E. BARRERA, M.D., AND L. M. KOPELOFF, PH.D.

NEW YORK

By means of chemical and immunologic agents, Kopeloff, Barrera and Kopeloff [1] produced in the monkey chronic recurrent convulsive seizures of the jacksonian and the generalized type. This was accomplished by exposing the cerebral motor cortex of the anesthetized monkey and inserting a small, round, laminated, compressed linen disk with a depression on one side containing an "effective substance" in such a manner as to maintain direct contact between this substance and the cortex. The meninges were sutured firmly over the disk to hold it in place, and the incision in the scalp was then closed by means of clips. In most of the animals in which seizures developed the attacks were first noted approximately three to eight weeks after operation.

PRESENT INVESTIGATION

Material and Methods.—In this communication we are reporting electroencephalographic observations on 5 monkeys, each weighing 3 to 4 kilograms, in which recurrent jacksonian seizures developed as a result of the unilateral application of aluminum hydroxide cream. Similarly, a study was made of a series of 10 control monkeys in which disks, applied to the motor cortex, were either empty or contained preparations which failed to induce seizures. Three additional monkeys not subjected to any operative procedure were employed as further controls.

Three pairs of electrodes were applied over the prefrontal, precentral and occipital regions, on corresponding areas on the two sides of the head, and simultaneous bilateral recordings were made. In some of the animals "indifferent" electrodes were placed over the mastoid process and on the ear lobe, but the use of these electrodes did not add materially to the information obtained with the six electrode placement already mentioned. Before each recording 1 cc. of sodium pentobarbital (0.75 grain [0.049 Gm.] per cubic centimeter) was administered to produce a mild narcotic state in the animal, half the dose being given intravenously and the other half intramuscularly. This procedure facilitated application of the electrodes, diminished or abolished violent movements of the animal and did not interfere appreciably with the final interpretation of the records with regard to bilateral differences. Records were taken approximately thirty minutes, one hour and one and one-half hours after injection of the drug. Most of the animals could be easily roused. When there was difficulty in waking a monkey, an initial record was taken, after which 0.5 to 1 cc. of metrazol was administered intramuscularly, and the recordings were continued. This procedure partially obviated the effects of the sodium pentobarbital by accelerating the return of a 14 to 22 cycle per second rhythm in place of the slow potentials produced by the narcotic state, but had relatively little effect on the abnormal activity resulting from the application of the aluminum hydroxide.

It should be stressed that in this particular study less interest was focused on the patterns of electrocortical activity in the monkey than on possible differences in activity between the two sides of the brain and on differences in animals (subjected to an operation) in which various substances were applied to either one or both motor cortices. Therefore, the routine use of drugs such as sodium pentobarbital and metrazol, regardless of their temporary effects on the pattern of the brain waves, did not seriously interfere with our final evaluation of significant bilateral differences or of notable bilateral abnormalities, since such changes were not encountered in the control animals. This observation seemed to be further borne out by comparison with the clinical picture.

Results.—Abnormal electroencephalographic activity was indicated by frequent delta waves and marked irregularities in the pattern. Ordinarily there was little difficulty in determining unilateral electroencephalographic abnormality. However, when the record exhibited bilateral symmetric activity in which irregular delta waves and sharp wave or spike potentials appeared out of all proportion to what might be expected in the normal monkey treated with pentobarbital and metrazol, a diffuse abnormality was suspected.

In general, a fairly close correlation existed between the clinical manifestations with respect to lateralization of the symptoms and the electroencephalographic pattern. In the monkeys showing seizures confined to, or predominating on, one side of the body, varying degrees of electroencephalographic abnormality were noted over the corresponding cerebral hemisphere. If the "effective" substance had been placed on the left motor cortex and a noneffective substance on the

Read at the Seventieth Annual Meeting of the American Neurological Association, New York, May 20, 1944.

From the Departments of Experimental Psychiatry and Bacteriology, New York State Psychiatric Institute and Hospital.

1. Kopeloff, L. M.; Barrera, S. E., and Kopeloff, N.: Recurrent Convulsive Seizures in Animals Produced by Immunologic and Chemical Means, Am. J. Psychiat. **98**:881 (May) 1942.

right motor area in the same monkey, with subsequent appearance of seizures on the right side, the left side of the cortex invariably showed a greater incidence of irregular slow potentials, usually associated with spike and sharp wave formations, than did the right side.

None of the animals with control preparations (noneffective substances) had seizures of any kind. The electroencephalograms of these animals showed delta activity, usually consisting of 4 to 6 cycles per second, about the site of operation, for only two or three weeks after operation. Then the incidence of these potentials diminished to a minimum or disappeared during the course of the next week or two. If the disk had been applied to but one motor area, a bilateral difference with respect to the incidence of potentials was noted for several weeks only. Subsequently, the electroencephalogram returned to normal (fig. 1). The 3 monkeys on which no operation was performed showed no electroencephalographic abnormalities.

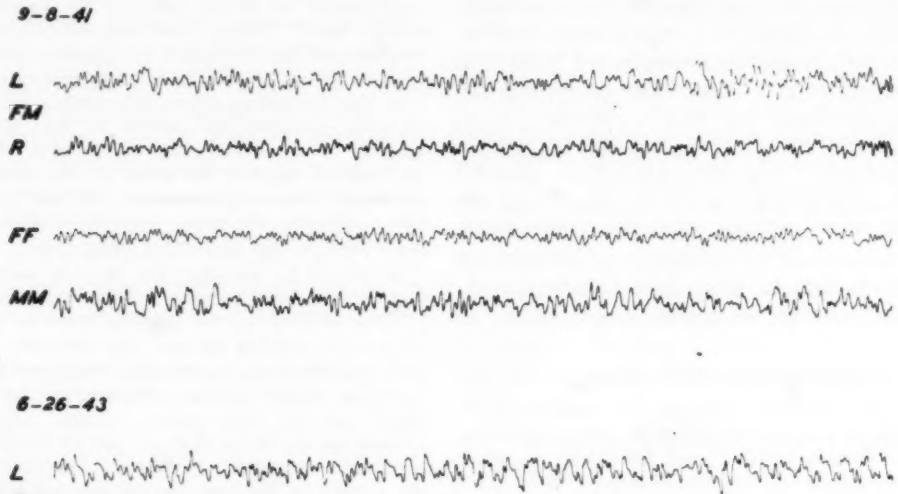


Fig. 1.—Electroencephalograms for monkey 351.

PROTOCOLS

Experimental Monkeys.—The protocols of 5 monkeys exhibiting clinical seizures over periods ranging from three months to more than two years will be presented in detail. During this period clinical and electroencephalographic observations were made at varying intervals. Electrical stimulation of the brain through the intact scalp was also employed to determine the threshold of excitability of the cortex. For this purpose we employed the commercial electric shock apparatus constructed by Mr. W. E. Rahm Jr. for clinical use. A flat metal electrode, measuring approximately 4 cm. in diameter, was firmly adjusted to the head over each temporal region, and the current was applied for one-tenth second. The clinical records include observations on "spontaneous" convulsive activity, or seizures precipitated by stimulation and excitation of the animal. The method of stimulation consisted of prodding the monkey with a stick and noisily striking the cage for about one minute. Pertinent details of each case follow.

MONKEY 351.—May 22, 1941: Operation: Aluminum hydroxide in disk applied directly over exposed left motor cerebral cortex in its medial superior aspect.

July 13: Generalized convulsive seizure observed for first time. Previously, neither clinical symptoms nor paresis.

July 16: Clonic seizure of right hand.

July 21: Generalized convulsion in forenoon and afternoon.

July 28: Generalized convulsion.

Sept. 8: Electroencephalogram: Some delta activity observed over left motor cortex, suggesting mild abnormality in this region.

Sept. 23: Generalized convulsion.

Sept. 30: Generalized convulsion, with predominance on right side.

Oct. 8: Clonic seizure of right arm and leg.

Oct. 20: Clonic seizure of right and left hands.

Oct. 29: Clonic seizure of right hand and leg; right side involved until November 5.

Nov. 5: Clonic seizure of left arm and leg; supination of right hand.

Nov. 6: Generalized convulsions until November 12.

Nov. 12: Clonic seizure of right arm and leg.

Nov. 14: Clonic seizure of right arm and leg and left hand.

Nov. 18: Generalized convulsion.

Nov. 18: Administration of phenobarbital sodium begun (0.5 to 2 grains [30 to 130 mg.] orally twice daily).

Nov. 19: Electrical stimulation: 50 volts: slow clonic seizure of right hand and arm for twenty seconds; 60 volts: clonic seizure of entire right side for fifteen seconds; 75 volts: generalized convulsion, beginning on right side (five seconds) and spreading to left side (total reaction, forty-two seconds).

Nov. 21-Dec. 13: No reaction on daily stimulation, except on December 1 and 3 (clonic seizures of right arm and leg).

Dec. 13: Phenobarbital therapy discontinued.

Dec. 15: Clonic seizure of right hand, right side of face and right leg, followed by delayed seizure of left hand.

Dec. 16: Generalized convulsion, beginning on left side.

Dec. 17, 18 and 20: Clonic seizures of arm, face and leg on the right side.

Dec. 24: Generalized convulsion.

Dec. 26-Jan. 23, 1942: No reaction following physical stimulation (ten times).

Feb. 11: Generalized convulsion.

Feb. 16: No reaction.

Feb. 19: Generalized convulsion.

March 6-May 6: No reaction following stimulation (five times).

May 14-27: Three generalized convulsions.

May 28-June 4: Five seizures of right side.

June 26: Electroencephalogram: Persistence and increase in degree of abnormality of focus in left motor area.

July 27-Jan. 22, 1943: No reaction following stimulation (six times).

Jan. 27: Generalized convulsion.

Feb. 8-July 1: No reaction (six times).

July 2: Electrical stimulation: 40 volts: clonic seizure of right hand; 55 volts: death.

In this animal it may be noted that the clinical picture, electroencephalographic observations and electrical stimulation of the head indicated a cerebral focus on the left side (fig. 1).

MONKEY 355.—May 23, 1941: Operation: Aluminum hydroxide applied over right motor cortex.

July 14: Clonic seizure of left arm and left side of face; focal activity continuing through November 6.

Oct. 20: Electroencephalogram: Definite focus in right motor cortex indicated by irregular delta waves and "sharp" activity over this region.

Nov. 10: Clonic seizure of left side, with first appearance of reaction in right arm.

Nov. 13: Clonic seizure of left side.

Neurologic examination: Slight clumsiness or "stiffness" of left limbs, with less spontaneous use and slight increase in muscular tone; knee jerk more active on left; reflexes of arms equal on two sides, with tendency to clench left hand.

Nov. 14: Clonic seizure of left side.

Nov. 14: Electrical stimulation: 50 volts: clonic seizure of left side, involving chiefly left hand, arm and eye (72 seconds); 60 volts: generalized convulsion.

Nov. 16: "Spontaneous" generalized convulsion observed for first time, beginning on left side.

Nov. 18: Clonic seizure of left side; twitches of right eyelid.

Nov. 21: Therapy with diphenylhydantoin sodium begun (0.5 to 30 grains [0.03 to 1.95 Gm.] orally, twice daily).

Nov. 21-Dec. 13: Clonic seizure of left side elicited daily.

Dec. 13: Diphenylhydantoin therapy discontinued.

Dec. 13-March 6, 1942: Clonic seizure of left side on stimulation on twenty-six occasions.

March 26: Clonic seizure of left side and right arm.

March 30-April 11: Clonic seizure of left side (four times).

April 17-Feb. 23, 1943: Generalized convulsions, starting on left side on stimulation on thirty occasions.

April 19: No reaction.

June 14: Generalized convulsion.

June 26: Electroencephalogram: Increased delta activity over right motor cortex and abnormal activity over left motor cortex; abnormality greater on right side.

July 6-Sept. 17: Ten stimulations: 4 generalized convulsions; 2 seizures on left side; 4 negative reactions.

July 24: Electroencephalogram: Record similar to that of June 26.

In this animal the presence of a focus on the right side was substantiated early (Nov. 14, 1941) by electrical stimulation, and on two occasions (June 26 and July 24, 1943) it was established by the electroencephalogram. Over a period, the electroencephalogram revealed an increase in the delta activity over the right motor cortex, as well as abnormal activity over the corresponding area on the left side. However, the right motor region showed relatively more abnormal activity than the left, indicating the persistence of a focus which, after two years, exhibited not only an increase in abnormal activity but a spread to the corresponding region in the opposite cortex, resulting in an apparent "mirror focus" (fig. 2). This is of particular interest since the "spread" could be correlated with the development of frequent, easily induced, generalized seizures.

MONKEY 364.—Oct. 8, 1941: Operation on left side: Normal rabbit serum in oxycholesterol-petrolatum ointment base applied to left motor cortex.

Oct. 20-Dec. 5: No reaction on stimulation (seven times).

Nov. 26 and Dec. 3: Electrical stimulation: Symmetric responses.

Dec. 10: Electroencephalogram: No significant differences between the two sides and no gross abnormal features.

Dec. 12-May 26, 1942: No reaction on stimulation (ten times).

June 24: Operation on right side: Aluminum hydroxide applied to right motor cortex.

July 21: 10 a. m.: clonic seizure of entire left side, followed immediately by clonic seizure of entire right side. 10:45 a. m., 11:15 a. m., 3:41 p. m.: generalized convulsions, beginning on left side. 3:40 p. m.: phenobarbital sodium, 1 grain (0.065 Gm.) injected subcutaneously.

July 22: Myoclonic contractions of left arm, 120 per minute.

July 21: No reaction.

Aug. 11: Electrical stimulation: 35 volts: Clonic seizure of leg, face and arm on left side.

Sept. 17: No reaction.

The application of noneffective material to the left motor cortex yielded no clinical reactions. The electroencephalogram showed no definite abnormal features and exhibited no significant bilateral differences (fig. 3). After electrical stimulation the responses were symmetric. However, after the application of an "effective" substance to the right motor cortex, the clinical

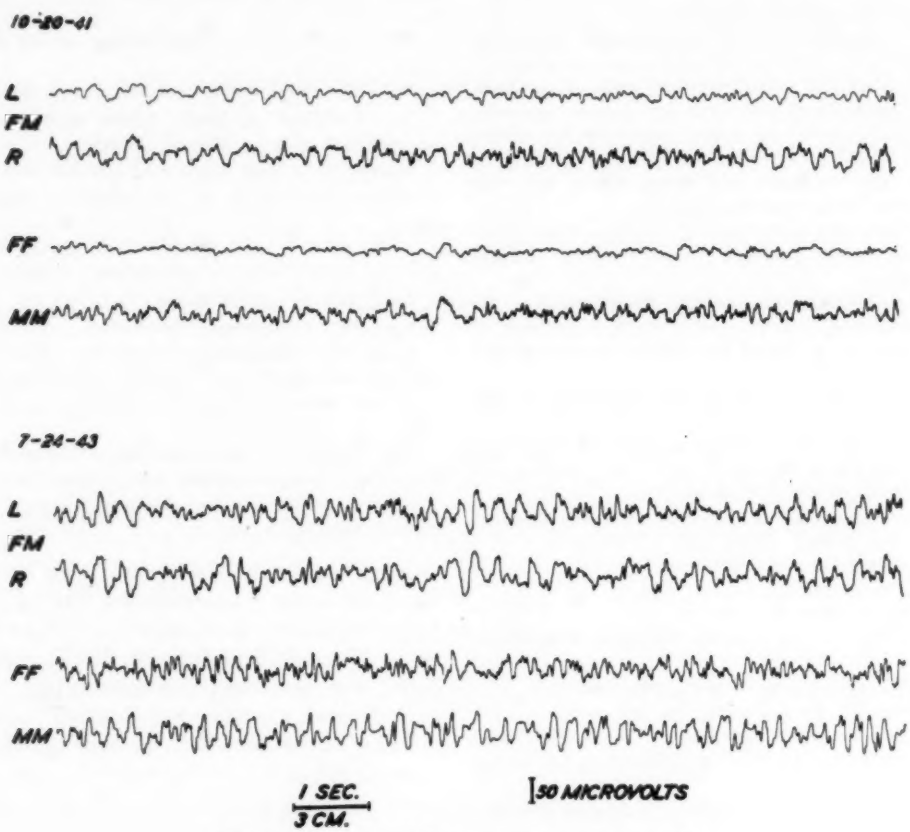


Fig. 2.—Electroencephalograms for monkey 355.

July 23-July 29: Myoclonic contractions of left arm, left side of face and left leg.

July 29: Protruding disk removed from right motor cortex.

Aug. 3; Aug. 23; Sept. 4: No reaction on stimulation.

Sept. 30: Myoclonic contractions of left leg.

Oct. 14-March 9, 1943: No reaction on five stimulations, except on Dec. 22, when myoclonic contractions of left leg appeared subsequently.

April 19: Clonic seizure of left side.

June 14: No reaction except for subsequent myoclonic contractions of left arm.

July 24: Electroencephalogram: Definite focus of abnormal activity over right motor cortex, consisting of much random, irregular delta activity and frequent spike formations.

manifestations were correlated with the electroencephalogram and the response to electrical stimulation, both of which indicated the presence of a definite focus of abnormal activity. It is of interest to note that this focus persisted after the disappearance of clinical seizures.

Monkey 383.—Nov. 5, 1941: Operation: Egg white in gelatin in disk applied to left motor cortex.

Nov. 13: Operation: Disk removed and replaced by disk containing egg white antiserum in oxycholesterol-petrolatum ointment base, in order to elicit possible anaphylactic response; no clinical reactions.

Nov. 14-26: No reaction (five stimulations).

Dec. 8: Electroencephalogram: No significant bilateral differences and no gross abnormal features.

Dec. 15-May 26, 1942: No reaction (eleven stimulations).

July 30: Operation: Aluminum hydroxide applied to right motor cortex.

Sept. 8-Dec. 11: Clonic seizure of left arm and left side of face (seven stimulations).

Dec. 22 and 30: No reaction.

Jan. 26, 1943: Clonic seizure of left side of face and left eyelid, arm and leg.

Feb. 18-June 14: No reaction (four stimulations).

June 29: Severe generalized convulsion.

June 30-July 1: No reaction.

July 10: Electroencephalogram: Abnormal focus over right motor area; relatively little abnormal activity over rest of cortex.

to the opposite motor cortex, a focus of abnormal activity was demonstrable by clinical observation, electroencephalographic changes and electrical stimulation.

MONKEY 384.—Nov. 5, 1941: Operation: Egg white and heat-killed tubercle bacilli in oxycholesterol-petrolatum ointment base applied over left motor cortex.

Nov. 14-May 26, 1942: No reaction (sixteen stimulations).

Aug. 3: Operation: Aluminum hydroxide applied over right motor cortex.

Sept. 16: Clonic seizure of left arm, left side of face and left leg, and then of right arm.

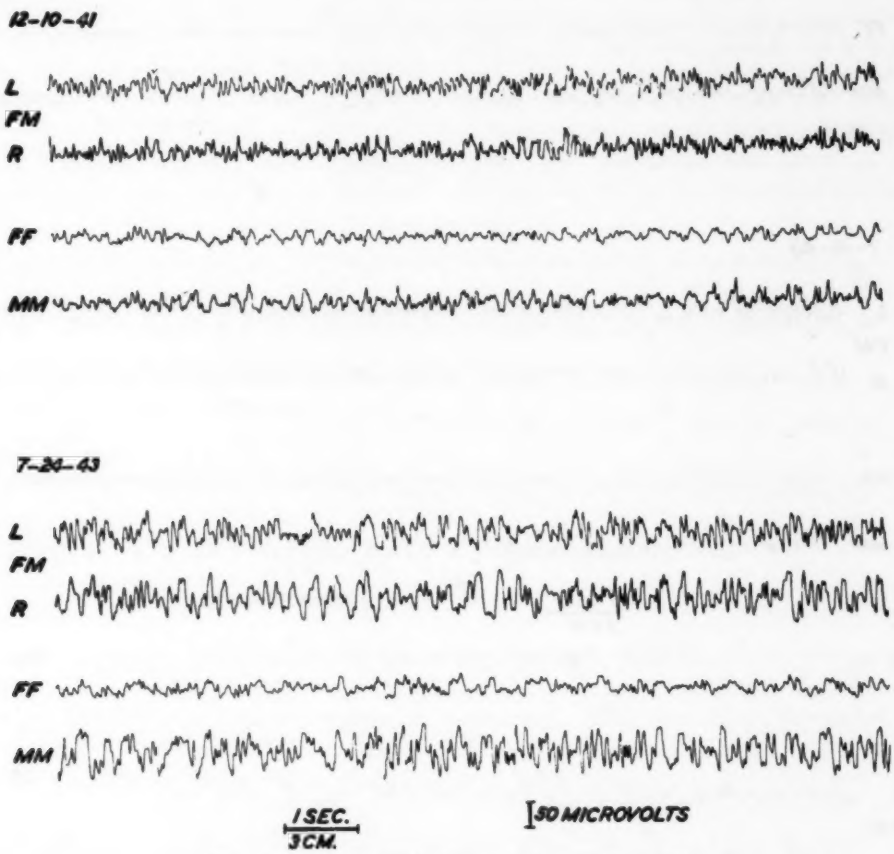


Fig. 3.—Electroencephalograms for monkey 364.

July 21: Severe generalized convulsion.

Aug. 9, 1943: Electrical stimulation: 50 volts: clonic seizure of left hand; 55 volts: generalized convulsion, beginning on left side. During latter part of general seizure, greater clonic excursions of left arm and left leg than of right limbs; immediately after convulsion, fine clonic movements of left thumb.

Sept. 17: No reaction.

This case serves as a suitable control for the operative procedure, since neither clinical nor significant electroencephalographic abnormalities followed the application of noneffective substances to one motor cortex (fig. 4). Moreover, when an "effective" substance was applied

Oct. 14; Nov. 6; Nov. 9: Clonic seizure of left arm, left side of face and left eyelid for forty seconds, and then of right arm, right side of face and right eyelid.

Nov. 23: Generalized convulsion beginning on left side.

Dec. 11: Generalized convulsion.

Dec. 22-Sept. 17, 1943: No reaction (twelve stimulations).

July 2: Electroencephalogram: focus of abnormal activity over right motor cortex, not as pronounced as in previous cases; at times equal and synchronous abnormal discharges from right and left motor area, with activity on right side for most part predominant.

Aug. 9: Electrical stimulation: 43 volts: slight clonic twitches of left arm; 65 volts: generalized convulsion, beginning in left arm.

It appears from these observations that after the disappearance of clinical symptoms for more than six months, electroencephalographic evidence of focal abnormality persisted (fig. 5).

The remaining 9 monkeys received control disks containing various noneffective substances over one motor area. In 2 of these 9 animals the disk was removed after one to two weeks and

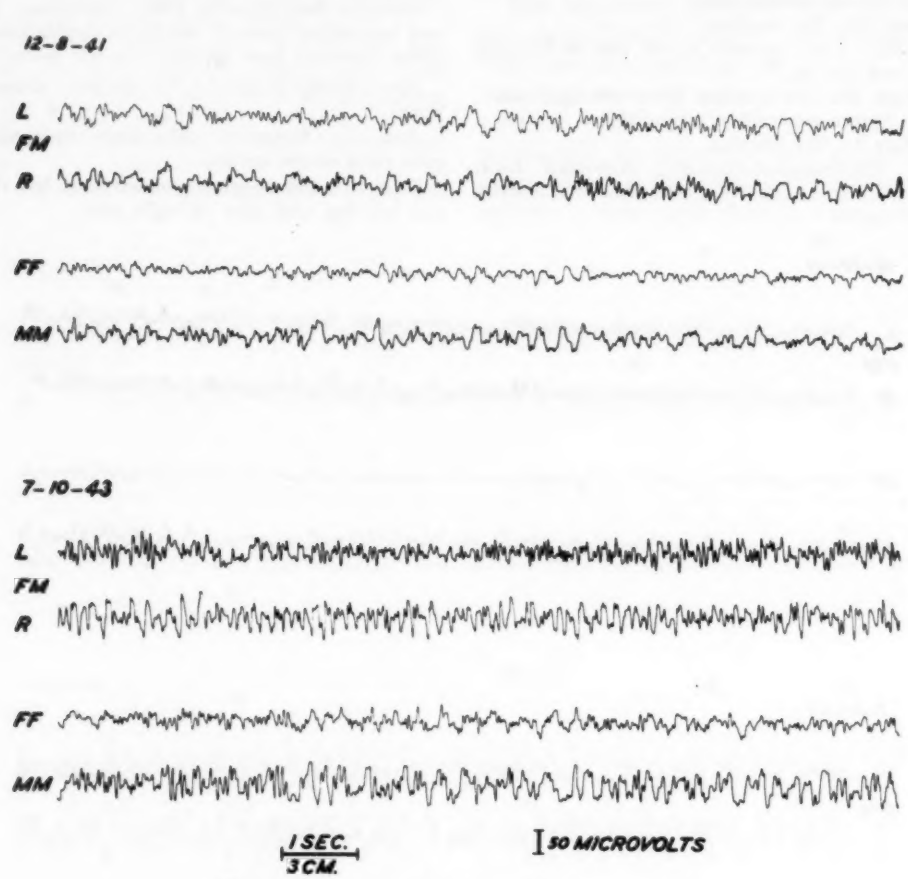


Fig. 4.—Electroencephalograms for monkey 383.

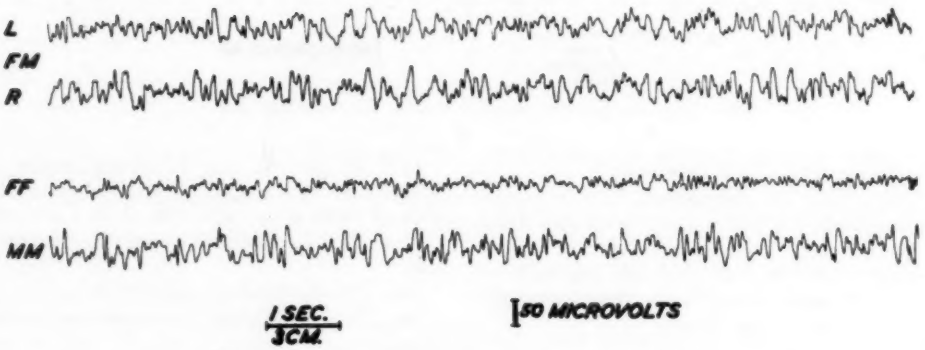


Fig. 5.—Electroencephalograms for monkey 384.

Control Monkeys.—A series of 13 monkeys served as controls. On 3 of these animals no operation was performed. In 1 monkey an empty disk was inserted over each motor area. replaced by a second control disk. The animals were stimulated and observed over a period of six to twelve months. Occasional electrical stimulation of 3 of these monkeys showed no focal

responses except in 1 animal, which, however, gave no focal response on several repetitions. Electroencephalograms revealed no persisting foci of abnormality subsequent to the acute postoperative phase and no significant differences in the two sides. A representative protocol of 1 of the monkeys follows (fig. 6).

Monkey 366.—Oct. 8, 1941: Operation: Disk containing rabbit serum in oxycholesterol-petrolatum ointment base applied over left motor cortex.

Nov. 26: Slight weakness in right arm.

Nov. 26; Dec. 3: Electrical stimulation: No focal responses.

Dec. 5 and 12: No convulsive seizures, either spontaneously or on stimulation.

Dec. 10: Electroencephalogram: No abnormal activity.

COMMENT

Elsewhere, Barrera, Kopeloff and Kopeloff [2] have presented histologic studies on a large series of monkeys treated with effective or noneffective substances. Disks containing these materials when applied to the motor regions of the cerebral cortex resulted either in typical seizures or in no symptoms whatever, the effect depending on the substance employed. The pathologic changes in monkeys exhibiting seizures could not be differentiated either in type or in degree from lesions in animals manifesting no symptoms. Although no histologic studies were carried out on the 5 monkeys reported on in this paper, inasmuch as they are still under observation, there is no reason to believe that the histopathologic changes would differ from those in the larger group previously studied. If subsequent histologic examination of these 5 animals bears out the previous observations, it would appear that a physiologic zone of hyperirritability, not demonstrable by histologic means, has been produced by the application of "active" agents to the cortex. The mere application of the disk to the cortex was not in itself sufficient to produce an electroencephalographic focus of abnormal activity for longer than two or three weeks, in spite of possible temporary local edema and changes in pressure. The absence of any clinical abnormalities was consistent with the electroencephalographic changes.

It should be noted that after the application of active material to the cortex there was an initial latent period of three to eight weeks during which the animal was free from seizures. In other monkeys studied during a corresponding period, the electroencephalogram indicated only mild abnormalities, chiefly of random delta activity, over the active disk. At the end of this "latent period," when unilateral muscular seizures first appeared, the focus of abnormal activity became more pronounced electroencephalographically. Subsequent to the period of unilateral response generalized convulsions usually occurred. This state lasted for variable periods, during which the unilateral response was often still predominant and the electroencephalogram still revealed a definite unilateral focus. The degree of abnormality could be correlated roughly with the severity of the clinical response. In the present study, in addition to similar observations just described, there was electroencephalographic evidence in 1 animal (monkey 355) that some spread of the original focus of abnormal potential had taken place to the opposite side during this period to produce an apparent "mirror focus."

In a monkey which exhibited seizures for three months, abnormal electroencephalographic activity still persisted after the disappearance of clinical seizures for seven months.

Further substantiation of the production of a focus of "hyperexcitability" was indicated by the application of an electrical stimulus to the fronto-

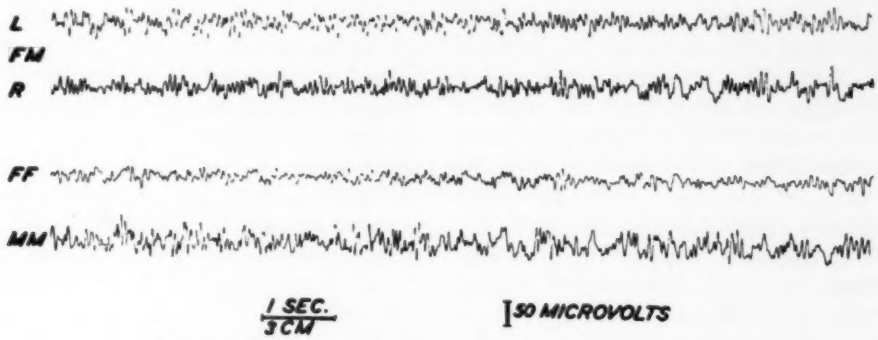


Fig. 6.—Electroencephalograms for monkey 366 (control).

2. Barrera, S. E.; Kopeloff, L. M., and Kopeloff, N.: Brain Lesions Associated with Experimental "Epileptiform" Seizures in the Monkey, Am. J. Psychiat. **7**: 727 (May) 1944.

temporal regions. "Threshold voltages" elicited unilateral responses which corresponded to the side of the cortex involved and were consistent with the electroencephalographic pattern.

SUMMARY

1. In a series of 5 monkeys an "effective" substance placed directly on the motor cortex resulted in the production of chronic jacksonian and generalized seizures for periods extending over two years after operation.

2. The presence of a unilateral focus for the discharge of abnormal potentials in the region of the applied disk was demonstrated in the electroencephalogram for many months after the operation. This focus corresponded with the clinical manifestations of seizures. From previous histopathologic studies of similar material, the foci did not appear to be the result of specific tissue damage but seemed to indicate an actual physiologic zone of hyperexcitability.

3. Electrical stimulation of the frontotemporal regions with "threshold voltages" produced unilateral responses which substantiated the clinical and electroencephalographic observations.

Miss Rhoda F. Bigelow and Miss Marguerite Manfreda gave technical assistance in obtaining the electroencephalographic tracings, and Mrs. Helen Kallman aided in preparing the figures.

722 West One Hundred and Sixty-Eighth Street.

GUIDE TO INTERVIEWING AND CLINICAL PERSONALITY STUDY

JOHN C. WHITEHORN, M.D.

BALTIMORE

LEARNING ABOUT THE PATIENT'S ATTITUDES

Human psychobiology, which is concerned with the integrated adaptive behavior of the human being, is of major importance in all fields of medicine. It is a fundamental purpose of psychiatric instruction and training to help the physician learn how to deal with the psychobiologic unit—the person—in action. The primary technical psychiatric procedure is the interview between the physician and the patient.

The aims and methods of psychiatric interviewing can be learned thoroughly only by experience, reflection and discussion with teachers, but a somewhat detailed discussion of the matter is offered here for preliminary guidance.

The universal aim of the physician at the beginning of the first interview with the patient in any case is to learn about the presenting problem, or chief complaint. Thereafter, aims diverge somewhat. One line of inquiry tries to answer the questions, "What noxious agent causes this patient to be ill; how does it do so, and how can it be eliminated?" Another line of inquiry concerns the patient himself, "What is his reaction to the noxious influence, or influences, and how can a knowledge of his personality be utilized in his care?" This study of the patient as a person acquires an enormous importance in certain instances from a surprising fact: The patient is often his own worst enemy. His attitudes are sometimes the most noxious agencies with which the physician has to contend.

The primary purpose of this guide is to help the physician to use the interview as a method of eliciting and evaluating patients' attitudes. It is not concerned primarily with the pathologic phenomena which should be noted in making a differential diagnosis. Its purpose is to aid the physician in the more difficult task of understanding the personality of the patient who is under study. Emotional reactions, which are expressions of the personality, play an important part in a wide range of medical and surgical conditions, not merely in psychotic or neurotic disturbances. Expertness in employment of the interview for this purpose is a universally useful medical skill. In the use of this guide in the development of skill in interviewing for the purpose of personality study, it is recommended that the student first read it over quickly for general orientation and then review various sections repeatedly in close relation to work with actual patients.

The clinical study of personality depends fundamentally on the physician's first hand study of the patient's attitudes. Sometimes the matter is left at the level of his getting a few opinions about the patient from relatives or other sources, but this information is inadequate, and often misleading.

To learn about the patient's attitudes is somewhat more difficult than to make a physical examination, largely because of two factors: First, the factual information sought is somewhat more subtle than that which the examiner seeks through inspection, palpation, percussion or auscultation; and, second, the technic involves the "personal equation" to a much more important degree. Indeed, one may truly say that the personal interaction between examiner and examinee constitutes the most vital part of this examination. The examiner must be doing two things—he participates in the interview and he observes at the same time. For purposes of logical discussion, these two roles may be separated, but in actual work they have to be combined.

ELEMENTARY STEPS

GETTING THE INTERVIEW STARTED

In learning to interview patients, one is inclined to depend on a prepared series of questions or topics, to "cover the ground" for one's superior; and it remains always a valuable aid to thoroughness to have in the back of one's mind, or in the back of one's desk, some scheme of systematic survey. The psychiatric textbooks give outlines which are particularly designed for the examination of psychotic patients, and special treatises on

From the Henry Phipps Psychiatric Clinic of the Johns Hopkins Hospital.

This guide has been developed as part of a presentation of the aims and methods of psychiatric work, used primarily in the training program for house officers and medical students.

this subject are available (Cheney [1] and Preu [2]). (In this clinic, the "Outline for Mental Status Examination" is provided, in mimeographed form, for use in examination of patients with psychiatric conditions, with many questions and suggestions arranged in systematic order.) Yet mere questioning, in an impersonal and completely unresponsive manner, tends to defeat the examiner's purpose of learning about the patient's attitudes, for patients do not talk revealingly to an interviewer who does not show signs of interest and of increasing understanding, that is, unless what the patient says seems to mean something to the interviewer. The patient's attitudes are not likely to appear at first, in answer to prepared questions, but later, in reaction to what he feels is the interviewer's response to his statements. Some response is needed to draw the patient out. The responses most helpful in eliciting significant statements are, in general, facial expressions and gestures—the ordinary human signs of interest and appreciation. The interviewer need not talk a great deal, and he surely does not need to begin forming and stating opinions. If he does have to talk a bit to get the patient started, he needs to be alert to what may be suggested thereby. Letting the patient tell his own story is the best way to avoid the troubles that arise from suggestibility. An attitude of alert interest and appreciation is the best means of avoiding the opposite extreme of having the patient "freeze up."

Furthermore, every interview has its psychotherapeutic or psychonoxious implications. It is incorrect to think that the examiner can first gather the information and then start the psychotherapy. The psychotherapy begins at the very first contact, in the sense of a mutual understanding and rapport. This does not mean that the interviewer has to start at once to offer advice and opinion—far from it.

THE PRESENTING PROBLEM AND THE PRESENT ILLNESS

With relatively few exceptions, interviews begin with the presenting problem, corresponding to the classic medical term "chief complaint." The presenting problem is likely to be the topic which provides the soundest basis for a conversation of mutual serious interest. Why did the patient come to the hospital, clinic or physician? What circumstances led to his coming at just this time? Had everything been going well up to this time?

The examiner should be sure to record verbatim the sentence in which the patient expresses his chief complaint. The manner in which this is stated generally indicates not only the chief complaint but the patient's attitude toward it, the record of which is lost if the complaint is translated into medical terms. Furthermore, all subsquent discussion is likely to revolve around this chief complaint; so it is a great help to be able to refer to it just as the patient stated it.

The story of the development of this chief complaint from the time the trouble began (the "present illness," as it is designated in the record) is usually the next topic to arise; here, again, the form of the patient's statement is important, although it is usually necessary to condense it for the record.

Some patients may say, "Well, doctor, you saw my record," or, "You saw my sister," or, "Do I have to go over all that again?" It may help, then, to say, "Yes, but we want most of all to know your story of the matter," or, "We need to understand how the situation has been affecting you—your life, your feelings, your activities or your welfare." It is of chief importance to get the patient's own formulation of the presenting problem. This will tell the physician a good deal about the patient himself, as well as about his sickness. The patient should also be encouraged to tell what he expects that the physician or the hospital can do about this presenting problem and what kind of help he is prepared to receive. Patients are seldom aware of all the resources of the medical profession. A patient who feels only moderately ill may say, "Well, doctor, maybe I need only a pill." Another, feeling gravely ill, may say, "Maybe I need an operation." Some patients find it difficult to accept the kind of help that can, and should, be given. So it is necessary to get an idea of what the patient expects and is ready for and the attitudes which must be met in gaining acceptance for the proper therapeutic measures.

SIMPLE TESTS OF MENTAL EFFICIENCY AND OBSERVATIONS ON MOOD

No general discussion of mental tests is given in this guide, since its primary purpose is to assist the interviewer in establishing rapport and in evaluating the dynamic features of the personality. A few simple suggestions are offered, however, which may serve as a means of quick orientation and as an aid in the tactful establishment of an acceptable basis for further mental

1. Cheney, C. O.: Outlines for Psychiatric Examinations, Utica, N. Y., State Hospital Press, 1934.

2. Preu, P. W.: Outline of Psychiatric Case-Study, ed. 2, New York, Paul B. Hoeber, Inc., 1943.

tests. These suggestions are of special importance when there is reason to think that the patient is psychotic.

After a certain portion of the interview has brought into discussion some of the patient's troubles, it may be appropriate to inquire, "While you have been anxious and uneasy about this condition, how has your work been going?" The answers to such a question will often reveal much more than the patient intends to say about his attitudes toward his responsibilities.

The examiner may then ask, "With this trouble on your mind, have you had difficulty in concentrating on your work?" Since nearly every one does at times have difficulty in concentrating, some kind of an affirmative reply is usually given, although perhaps vague and qualified. Then the interviewer can appropriately say, "Well, I have a little test for concentration which I would like you to do, in the way of mental arithmetic. First, subtract 7 from 100." When the answer has been given as 93, say, "Now subtract 7 from that." When that answer has been given, say, "Now, go right on subtracting 7 each time, without my interrupting you." The examiner does not interrupt except to nod or to say "Go on," unless the patient is floundering badly. If the patient has done well (all the answers having been given correctly, within one half or three quarters of a minute), the examiner may ask, "Is that the correct remainder?" It should be noted whether the patient checks his work by dividing 100 by 7.[3]

In accordance with the patient's ability to keep oriented to the task at hand through this performance, the examiner may then inquire appropriately about general orientation, e. g., "Do you have any (or similar) difficulty in concentrating on practical matters, such as keeping track of current events—of time?—of your meals?—of people's names?—of where you are?" If he is uncertain, he should be asked for specific answers.

The presentation of this series of tasks to the patient as having to do with his ability to concentrate is psychologically reasonable. It has, also, the advantage of permitting efforts and failures without severe loss of self esteem, since every one can admit difficulty in concentrating at times, whereas he might feel insulted by the bald questions.

Appropriate situations may occur during these "concentration" tasks for the question, "When you have difficulties in concentrating, are you distracted by other thoughts?—by noises?—by imaginations?" Then the examiner may ask, rather vaguely, "Have you had any strange or unusual psychic experiences?" or, more bluntly, "Do you hear voices—or see visions—or receive messages—in uncommon ways?" These suggestions afford a minimal scrutiny of the more outstanding psychotic symptoms if present.

Observations on general behavior and the style and tempo of talk and activity should be noted; they may form the basis for the evaluation of such moods as sadness, fear and elation. Disorders of mood are usually reflected, also, in disturbances of sleep, appetite and bowel function.

DEPRESSED STATES AND THEIR INFLUENCE ON THE INTERVIEW

When the patient is found to be in a sad mood or is gloomily irritable, consideration should be shown to his thinking difficulty. An apparently simple conversation may be overwhelmingly difficult for the retarded patient. It is the moderately or mildly depressed patient who is most likely to have this feature of his condition overlooked, and who therefore runs the greatest risk of being exhausted by an unduly complex or prolonged interview. An exhausted depressed patient may be a source of much misinformation, either because he misunderstands the context of a discussion or because, in order to be spared further effort he may answer "yes" to any question or, in an undiscriminating way, disagree with everything.

ROUTINE QUESTIONS AND EXAMINATIONS

The discussion of the chief complaint and the present illness will often bring spontaneous statements on many of the points of information needed for the medical record, but some points will usually be left untouched. When a measure of rapport and mutual understanding has been reached through some minutes of rather informal and spontaneous interviewing, the examiner will usually find it acceptable to change the pace and style of the interview, in order to complete the routine information. He may say, "Now I have some specific questions to ask you about your past life. When and where were you born? What illnesses or operations have you had? Where and when?" and so on, through the usual simple questions about school, jobs, marriage and children and social and economic circumstances, including, in a matter-of-fact way, questions about habits (use of alcohol and tobacco and sex experience). The patient may be asked for a simple self evaluation of his personality traits (whether he is reserved, moody, tense, excitable

3. Hayman, M.: Two Minute Clinical Test for Measurement of Intellectual Impairment in Psychiatric Disorders, Arch. Neurol. & Psychiat. **47**:454 (March) 1942.

or steady). With regard to the family history, the examiner may at first quickly review the ages and general health of the parents and siblings and the occurrence of any illnesses among them comparable in the patient's eyes to his own condition, and later expand the inquiry as needed for the biographic understanding of family relationships. Information from other persons may be required to complete, supplement or check the patient's account.

For an adequate personality study, the biography of the patient is, next to actual interview and observation, the most important source of information. The art of biography for medical purposes is not specifically discussed in this guide, but later sections deal with many of the principles involved in it.

SPECIAL ASPECTS OF INTERVIEWING

COMMON DIFFICULTIES IN PSYCHIATRIC INTERVIEWING

The examiner should avoid getting into unprofitable arguments with a patient. Some patients, especially those with a chronic, obscure disease or a neurosis, may put an uncomplimentary interpretation on any evidence of interest in their personal reactions, saying "So you thing I'm abnormal, do you?" or, "Do you think I am only imagining these things?" Such remarks may at times be turned to advantage by asking, in turn, "What suggested that to you?" Whereupon the patient may tell of relatives, friends or physicians who have "accused" him of being neurotic. The physician's quizzical eyebrow or his repetition in a questioning tone of the word "accused" may then elicit a revealing rush of self-defensive remarks about "misunderstandings" over what some one may have called "imaginary" illness or over a lack of appreciation of the patient's trouble.

Sometimes a patient may sink or retire into a bog of tearful self pity or self defensiveness and need a bit of assistance in climbing out or a bit of stimulation. Restraint and discretion should be exercised in order not to overdo it.

If the examiner has already picked up some facts of encouraging or appreciative potentialities, he can bring this knowledge into play at this point. He may say, for example, "But, in spite of these difficulties and misunderstandings, I understood you had kept active in the care of your home (or in church duties, or looked after your sister's child, or were secretary of your club)."

It is well to be specific in such remarks, as in any questions concerning successes and achievement. A generalized question about such matters can be disheartening to one who cannot at the moment recall any achievements.

Sometimes a patient may stick on the issue that the physician thinks him abnormal. If it is not possible to get around this point unobtrusively, the examiner may say something like this, "I had not thought particularly that you were 'abnormal' (or 'imagining things' or 'neurotic,' or whatever point the patient has raised) but just that you are human, and as a doctor I am of course concerned about how your human interests and activities affect your illness and your health. It is a necessary part of my business to learn about such matters."

The experienced interviewer will sometimes find it an advantage to acknowledge promptly, if it is true, that the patient's behavior or remarks have raised in his mind the possibility that the patient has become seriously handicapped by depression or has been confused or is jumping to conclusions too quickly, and will remark, "Well, you do seem depressed," or, "What you have just said sounds very strange to me," or, "Well, maybe so, but tell me more, so I can better understand your difficulty."

"IRRELEVANT TALK" AND ITS REVELANCE FOR PERSONALITY PROBLEMS

Some patients do not need such careful maneuvering to get them talking about their sickness and themselves. Indeed, some physicians who are untrained in using the interview for personality study complain at times of the patient who talks too much, who "hands out just a lot of circumstantial and irrelevant talk." If one is studying the personality of a patient, there is no such thing as "irrelevant talk." The irrelevance is merely a condition of the interviewer's mind—he doesn't know what to make of it. The observer may listen, out of politeness, or because he has been told there is psychotherapeutic value in letting the patient talk, but he may not know what to listen for; in that case, the psychotherapeutic value may not be very great, for the patient may sense that no progress is being made.

The inexperienced interviewer may wonder how he can get the facts required for a respectable case record while letting the patient ramble. It is sometimes necessary to sacrifice the value of an unobstructed statement in order to hasten along to some other task. Actually, however, much of the information needed will come out spontaneously, and opportunities will also arise spontaneously for the necessary factual questions, such as those concerned with ages and names and the relationships of parents, siblings and others. The interviewer will often find it distinctly helpful to pick up promptly the familiar names of persons as they are spontaneously men-

tioned. A relatively few seconds of intense effort, applied successfully at the first opportunity, in learning names and chronologic sequences may gain him a rapport with the patient which is worth hours of subsequent effort.

Without getting into deeper meanings at this stage, I may say that the patient's "irrelevant talk" usually means that he feels the need of self justification, for some reason or other. To discover the "some reason or other" may be sometimes of much greater medical importance than anything else the physician can do. He should not, therefore, merely wait for the talk to subside or impatiently brush it aside. He should get down at least a few sentences for careful study. The interviewer does well, in general, to keep his mouth shut and his mind alert to all evidences from which he can infer the nature of the implied accusation or guilt against which the "irrelevant talk" or behavior constitutes a defense. He has the opportunity to learn in this way what "it is all about," by a process somewhat analogous to the surveyor's triangulation, thus: He may take two statements of the patient and imaginatively construct a statement to which they could be the logical replies. If a considerable number of these construction lines converge toward the same point, the interviewer thus acquires a tentative idea as to what the talk is all "about." He may even throw out tentative questions or remarks to check his inferences. It is not recommended to seize the patient, so to speak, and make him face this central issue. In whatever the interviewer says he should keep to the same tangential lines which the patient has already laid down and use so far as possible words which the patient has used. Skill in such "triangulation" is gained by close attention to detail and careful review of recorded interviews.

THE INTERVIEW AS A CONSTRUCTIVE EXPERIENCE FOR THE PATIENT

The aim of this guide is primarily to aid the student of medicine as clinical clerk or intern in the task which to him is paramount. That task is, primarily, the diagnostic responsibility of arriving at a sound knowledge of the patient and an insight into his condition and reactions. Nevertheless, it should be clearly recognized that the interview between patient and physician may have meanings for the patient which are quite different. Why should any patient wish to carry on an interview?

The student's common sense tells him that the patient should wish to cooperate in order to establish a correct diagnosis, and the patient may, of course, acknowledge the logic of this proposition and yet be impatient, and a bit emotionally resistant, about what may seem unnecessary personal exposures.

The most general and reliable incentive for the patient's participation in interviews is the desire for understanding and an appreciative response. Frequently this desire is a stronger motive than is the desire to cooperate with complete truthfulness for the purpose of getting relief from pain or other symptoms of illness. The physician comes to expect a certain amount of falsification and deceit, as automatic human reactions, not to be viewed with too much indignation. The "rate of discount" varies with the patient and, also, with the rapport. When the interviewer senses keenly the patient's need for understanding, it is a common error to say hastily and reassuringly, "Yes, I understand," when, in fact, he does not understand. Such empty reassurance defeats its own purpose and blocks further progress. Real understanding, useful to the patient, is best shown in appreciative listening, with brief comments and questions geared closely and simply to the theme of the patient's preoccupations. Courtesy and respect shown in this way need not excessively prolong the interview; in fact, the session is often thereby speeded up and brought around to certain objectives of the interviewer much more promptly and effectively than by his keeping the patient crowded to the wall by a barrage of questions.

In any interview which invites spontaneity, the person interviewed may make constructive use of the opportunity to gain a better perspective and orientation. In therapeutic interviews this opportunity is systematically developed and used. Through the respectful, but candid, interaction between the attitudes of the two participants knotty problems may be untangled with much greater effectiveness than in any purely logical analysis. In this sort of work the interviewer needs to have a fairly sound understanding of his own attitudes as they bear on the patient's problem, lest he impose on the patient new difficulties.

THE IMMEDIATE SITUATION AND THE PATIENT'S ATTITUDE TOWARD IT

The foregoing suggestions have been formulated primarily as aids to the physician in starting an interview with a new patient in the outpatient department. If the patient comes to the interview from a psychiatric ward, or is interviewed right in the ward, there will of course be a somewhat different set of expectations to deal with. The patient may be guardedly self defensive, or he may be

openly self assertive, with delusional material prominently thrust into the foreground; and the interviewer may be blinded to the real opportunity for understanding because of prejudiced or inadequate conceptions of what such behavior means. On the other hand, if the patient is in a surgical or an obstetric service, both he and the interviewer are likely to have quite another sort of expectations, and may perhaps be temporarily blind to the importance of emotional factors.

Even if informed by an instructor in advance that the patient's condition has been classified in a certain diagnostic category, the interviewer has still a natural opportunity to inquire about the individual patient's manner of experiencing this illness in his own individual way and his manner of meeting the problems presented by the illness. There is always, therefore, some appropriate route by which to open a pertinent discussion of personal issues, if only ingenuity and tact are skilfully utilized.

The patient usually finds himself, by reason of his illness—whatever it may be—in a state of unaccustomed helplessness. How does he take this? He may be chagrined or embarrassed about it and strive to minimize his helplessness, or he may welcome the invalid state. The manner in which a patient, in the situation of dependency imposed by illness, accepts or solicits sympathetic consideration may provide ready clues to his aptitude for the invalid role. In some persons this aptitude for invalidism is great indeed at all times, and it may be the main difficulty with which the physician has to contend. Or it may be that a tendency to welcome invalidism may represent only the need to escape from a temporary, but unusually harassing, set of anxieties. The physician's awareness of the need to evaluate such a factor will ordinarily lead to inquiries as to the patient's responsibilities. At the same time, he will feel the need to frame the discussion in terms acceptable to the patient, and so will find himself discussing occupational fatigue or point rationing or the children's school difficulties, not as irrelevant intrusions in the history taking, or as separate and unrelated items, but as integrally related and medically significant affairs, pertinent to the evaluation of the patient's capacity to tolerate responsibility and anxiety.

The physician's preparedness to gain significant insight into the patient's needs and difficulties through discussion of the patient's anxieties depends largely on his understanding of the psychologic and physiologic function of anxiety and the various human reactions and defenses against anxiety. For this reason, these suggestions for interviewing are supplemented here by a review of the dynamic considerations involved in dealing with anxiety.

SUMMARY

In a brief summary of all the preceding elementary discussions, it may be said that, in whatever conditions the personality of the patient is considered to constitute an important factor in the causation or the management of his illness, the examiner should seek, preferably through interviews with the patient himself, to establish the following points: (1) the patient's own spontaneous statement of his presenting problem (or "chief complaint"); (2) the special pressures in the patient's situation—personal, economic and social (including legal, political and military factors)—and the patient's attitudes thereto; (3) the way in which the patient's responsibilities or anxieties are increased or decreased by the illness; (4) the patient's general predisposition or resistance to the invalid state, and (5) the attitude of the patient to the physician, the hospital, etc.

DYNAMIC CONSIDERATIONS IN PERSONALITY STUDY

The preceding suggestions for the interview of patients cover only the most elementary aspects. For more penetrating and significant progress in clinical personality study, one cannot well depend on preformulated detailed suggestions. It is more helpful in the long run to have a clearly formulated conception of the varieties of ways in which the medically important features of personality are developed and come to expression. Good descriptions of the observable patterns of various personality disorders are available in all the modern textbooks of psychiatry. Many of these manifestations show clearly in direct reactions to the interview and the examination by the physician; other features come to light in the review of the patient's life. In the preparation of this guide, it is assumed that the user has available a good descriptive textbook and clinical cases for examination and study. Since this guide is oriented toward therapeutic aims, the considerations presented here are shaped to help the physician in getting a grasp of the dynamic personal forces manifested in the clinical phenomena. The viewpoint here is functional, rather than symptomatic. The aim is to help the examiner in utilizing the interview to gain an understanding of how the patient has been reduced to a second rate mode of adjustment and why he may be inclined to remain slumped in a relatively maladjusted state, rather

than to endure the distress and anxiety involved in risking attempts at a better personal adjustment. Such an understanding, if obtainable, enables the physician to fit the therapeutic strategy to the situation and the person.

In the focusing of attention on the biographic development of personality trends, the user of this guide is cautioned against the tempting fallacy of attributing the sole cause of an episode of illness to any single dramatic personal event or impersonal trauma. It is wise to remember how intricately the integrative mechanisms operate in bringing a multiplicity of influences to bear on behavior. Reaction is determined both by attitude and by circumstance, and in the shaping of attitudes single events become significant through relation to the whole course of the personality development.

RELATIVELY SIMPLE REACTIONS OF ANXIETY

ANXIETY; ITS EFFECTS AND ANTIDOTES

Anxiety—the condition of heightened tension, usually associated with fear or excitement—is a universal human experience of great biologic importance, both for good and for ill.

On the good side, anxiety mobilizes physiologic mechanisms for violent activity and puts one's faculties "on the alert." Anxiety may even do some good at times in the very respect in which its effects seem worst, that is, in interrupting and disorganizing a pattern of action, for this disorganization of a pattern may jar one out of a rut unsuited to the demands of the moment and open up the possibility of more appropriate behavior and attitudes. Whether or not such a new possibility will be realized will depend of course on other features of the personality and on the situation. Within limits, and when concerned with issues which are tolerable to one, such disturbing events may serve as useful stimulants to the growth of personality.

On the bad side, when anxiety is excessive or too prolonged, it may too thoroughly interrupt and disorganize one. The physiologic mobilization in anxiety, if prolonged and unused, may expend one's resources and seriously disrupt the automatic regulation of the body, particularly in the gastrointestinal, circulatory and respiratory systems.

The natural antidote to anxiety is action—definite and specific action, designed to overcome or eliminate the source of anxiety. The relief from anxiety through action is not usually delayed until the source is overcome. Vigorous and aggressive action may itself dispel anxiety, sometimes almost in a flash. Hence the desirability for soldiers of thorough training and drill, so that action may be prompt and vigorous. By providing release through action, one may, if the action is appropriate, make an asset out of the increased tension of the anxious state, rather than permit it to blow up into demoralization.

Because action in itself, even relatively futile action, has some value in reducing anxiety, many persons drift into habits of resorting to certain set activities when anxious—activities which range in complexity from simple tics or an embarrassed laugh or twiddling a watch charm to the most elaborate obsessive rituals.

In the course of the life experience, particularly in early childhood and in adolescence, the anxiety and tension associated with fear and excitement may become associated, almost as in the conditioned reflex experiment, with specific emotional and visceral reactions, such as disgust, vomiting, loss of appetite and trigger-like reactions of the sexual system to specific stimuli.

Common examples of such combinations of behavioral and visceral disorders, associated with anxiety, are found in fastidiousness toward food, easy disgustability, dizziness, and even amenorrhea, as illustrated in anorexia nervosa. Excessive motor and secretory reactions of the stomach or colon are also frequently caught up in such associated patterns, with rather specific emotional connotations and in correlation with definite personality trends. There is more than merely verbal similarity in defecatory and desecratory impulses.

Physicians, because of their professional preoccupation with problems of life and death, are apt to assume that the fear of death is the great common denominator of anxiety. This is probably not true. Even in deadly warfare one's greatest apprehension is not of death but of being maimed or of failing in one's duty, and that, in large part, because one dreads the reactions of other persons. In common clinical experience, when the physician has to deal with recurrent states of anxiety, or with certain second rate, self-defensive reactions habitually utilized for minimizing anxiety, it is most commonly found that the anxieties arise out of interpersonal relationships and interpersonal attitudes. Even financial worries are not concerned simply with money—they have to do with one's obligations and responsibilities to other persons and the attitudes of other persons.

People differ enormously in their capacity to tolerate anxiety. Some recoil from almost any anxiety situation; others accept an anxiety experience as a necessary feature of real living and "sweat it out" as well as they can. For many persons, anxiety and its immediate and remote

repercussions disturb physiologic functions to a degree which may be experienced as an illness, or in a manner which seriously complicates other illnesses. Particularly for those not endowed with abundant vitality and enthusiasm, recurrent anxiety and tension may spell the difference between fairly good health and incapacitating illness.

SIMPLER PHYSIOLOGIC EXPRESSIONS OF ANXIETY, THEIR DETECTION AND TREATMENT

A considerable number of persons who come for medical study and treatment present symptoms which are nothing more than the regular physiologic expressions of anxiety. With the inevitable tendency to self diagnosis, these symptoms may have been formulated by the patient as "stomach trouble" or "heart trouble," and through introspective exaggeration and hearsay these self-diagnosed conditions may be supplied with an additional layer of suggested signs and symptoms. It may require much examination and testing to rule out such plausible misunderstandings.

The initial interview gives an opportunity for the observation of simple and useful signs pertinent to the detection and evaluation of anxiety, such as moist hands, mopping of sweat from the forehead, tense postures, fidgety movements of the hands or feet, uneven or strained voice, swallowing movements, wide pupils, excessive vigilance or preoccupied inattentiveness and general restlessness, with their topically determined fluctuations.

The treatment of wet palms, tightness in the throat or tachycardia which originates in anxiety proceeds effectively, not by the application of remedies to the palms, the throat or the heart, or, indeed, by the application of remedies at all, but by the resolution of the sources of anxiety.

The same principle should be borne in mind in dealing with the more complicated neurotic mechanisms. Therapeutic strategy is better directed toward helping the patient deal effectively with more fundamental difficulties—usually difficulties in personal adjustment.

With a certain proportion of patients the whole business of psychiatric interviewing and personality study—and of treatment also—may be comprehended in a fairly simple interview. The patient may be given some authoritative help in understanding that his condition has developed in part through the results of anxiety, and he may be aided in a simple way in focusing his capacities on a proper solution of the problem which is responsible for the anxiety. If the patient can then promptly and effectively deal with the cause of anxiety, this may be all the psychiatric interviewing that is necessary to help him to the cure of his physiologic upset.

HINDRANCES TO THE SIMPLE MANAGEMENT OF ANXIETY EFFECTS

Unfortunately, simple enlightenment about anxiety may not be enough to get the patient on the road to health. This may be because the anxiety-producing situation is in itself too difficult for the patient to solve, or because certain attitudes of the patient hinder his attempts at its solution, or because the patient's anxieties actually arise out of certain attitudes which reproduce the same sort of situation again and again. Under such circumstances the psychiatric interview has to reach another level of subtlety. In general, it is foolish to be more subtle than is necessary. Many of the subtleties of psychiatric interviewing are required only in the most difficult cases, suitable only for the highly trained specialist, but there is a considerable range of mild forms of personality disorder, involving only a moderate complexity of the personality functions, which can be disentangled, helpfully formulated and greatly relieved through a relatively modest understanding of certain common self-defensive reaction patterns.

NEUROTIC DEFENSES AND THEIR RELATION TO EVERYDAY LIVING

COMMON DEFENSES AGAINST ANXIETY AND THEIR ILL EFFECTS

It is a common observation that chronic tension or acute anxiety arises from dissatisfaction and that dissatisfaction comes from the nonsuccess of one's undertakings. As a rough approximation to the truth, it may be said that success brings satisfaction and failure brings dissatisfaction. But without aspiration or ambition there is no keen experience of failure. The formula for satisfaction might be expressed better as a ratio—the ratio of achievement to aspiration. Even great achievement may leave great dissatisfaction if it falls too far short of one's aspirations. This formula is not quite adequate, either, because it implies, mathematically, that great achievement with slight aspiration should give huge satisfaction, which is not altogether true. But the principal reason for the inadequacy of such simple formulas for satisfaction or dissatisfaction lies in their failure to take into account the great importance of one's emotional relationships with other persons, from which, in fact, arise the greater part of one's satisfactions or dissatisfactions. An adequate statement must include such considerations. To include them, it may be said that satisfaction, in human life, is gained through successful self expression in

a manner which maintains emotional security and self assurance through the affection and respect of others.

This complicated formula is not fulfilled for any one 100 per cent of the time. Some persons, emotionally insecure, find themselves regularly inhibited in self expression because of paralyzing uncertainty as to the affection and respect in which they are held, but may burst out spasmodically at times in awkward and ungracious self assertion; others, overprotected and overindulged, may acquire offensive habits of self assertion through lack of an appropriate milieu for the development of their responsiveness to the feelings of others.

The failure, in any given event, to meet all the conditions of this formula for satisfaction commonly produces anxiety or depression. Such anxiety may be a useful experience, spurring one to better effort next time. On the other hand, depression may, also, be a helpful experience, in leading to a more temperate exertion and a more moderate aim. But either anxiety or depression, or a combination of the two, is unhealthy if it is so excessive or prolonged as to prevent integrated effort. Such pathologic anxiety or depression may be called primary psychopathology, for convenience of discussion, to assist in the consideration of secondary psychopathologic reactions, namely, certain neurotic defenses against depression and anxiety. These are psychobiologic devices which protect one's self esteem in second rate fashion—a sort of automatic avenue to consolation. Perhaps the simplest of these automatic face-saving maneuvers are the hypochondriacal and neurasthenic complaints, sickness and fatigue being almost universally acceptable excuses for nonperformance, as well as means for gaining attention and pity. Attention and pity are desirable interpersonal supports to emotional security—not so good as respect and affection, but still worth while if one cannot have a full measure of the more desirable responses. (In later sections some of these neurotic defenses will be briefly discussed.)

From the medical point of view, the neurotic defense reactions have two undesirable features: First, they are, so to speak, habit forming; that is, they dull the psychic pain of failure or insecurity, with considerable substitutive gratification, and so tend to be repeated again and again; second, they are not altogether satisfying, since they do not provide good support, from the emotional responses of others, to one's feeling of self assurance, but tend to produce complications of shame, disgust, resentment, dependence, rebellion and other mixed feelings, which, again, stir up anxiety or depression and so establish a kind of vicious circle, like the drunkard's remorse, which can be drowned only in further alcoholic oblivion.

Two elementary principles of psychotherapeutic treatment follow directly from these considerations and may conveniently be mentioned here: First, it is desirable to help the neurotic patient rediscover and utilize capacities and interests the expression of which may lead to more genuine gratification; and, second, in some of the more difficult cases the physician may have to carry out a more fundamental procedure by going back to items of "unfinished business," in which the neurotic pattern was resorted to, and helping the patient complete the business in a more permanently successful and gratifying way, compatible with a reasonably high measure of self esteem, and therefore not requiring neurotic self defense.

A third principle must also be carried in mind in any attempt at the more radical treatment of neurotic persons, namely, that the neurotic defenses, troublesome and hindering as they may be, have considerable value in maintaining some kind of stability, and the therapeutist should not attempt to blast them away without careful provision of other supportive measures and thoughtful consideration of the patient's alternative modes of adjustment. It is probably fortunate that neurotic adjustments are rather resistant to change. Otherwise, overenthusiastic psychotherapeutists, lacking in wisdom, might do more harm than good.

"COMPENSATED" AND "DECOMPENSATED" NEUROSES

In clinical medicine one finds not infrequently that a seriously crippled heart may nevertheless perform adequately its necessary functions. The heart is then said to be compensated. The person may carry on at a somewhat reduced level of activity without distress. In a somewhat comparable way, the neurotic person may have developed an adjustment to life—maladjustment though it may be—which enables him to carry on without great distress. Obsessive scrupulosity is a common form of such adjustment, whereby anxiety or feelings of guilt are kept at a minimum. The hypochondriacal domination of a household may, likewise, keep one "compensated" in this sense. On occasions which threaten such adjustments, or which call heavily on one's capacities for interpersonal relationships, such "compensation" may break and a state of considerable anxiety develop. In a figurative way, this condition may be characterized as a "decompensated neurosis." Since it is usually just such a "decompensated" state which drives one to

the physician for help, most neurotic persons who come to the physician's attention show anxiety, in addition to the other neurotic phenomena.

A difficult therapeutic dilemma not infrequently arises therefrom, which perplexes even the most experienced physician—whether to be content with efforts to assuage the anxiety or to try, more ambitiously, to help the patient toward a more radical readjustment. If the patient cannot endure much anxiety, attempts at a radical readjustment are likely to be defeated.

PERSONALITY TRENDS

HABITUAL RESPONSES TO CONFLICT AND ANXIETY

All persons, in the process of developing personality, have found and acquired certain aptitudes for dealing with anxiety-producing situations and persons, and these habits are the expressions of attitudes and sentiments which may be called personality trends. The regularity and adequacy of these trends determine largely the effectiveness and satisfaction in a personal adjustment to life. Abnormal personality trends have received more study than normal trends; yet this contrast is not so great as one might think because of the fact that the abnormal trend is often but an exaggerated or unbalanced expression of the normal one.

Such considerations, unnecessary perhaps in dealing with relatively simple reactions to real and immediate situations, become of paramount importance in the more serious neurotic and psychotic reaction patterns. As has previously been indicated, a given patient's illness may represent in large measure the development of an automatic defense mechanism out of a personality trend, by which he gains consolation at the price of moderate distress or disability and thus saves himself from an intolerable anxiety or tension.

In interviews with the patient, it is a good habit to scrutinize his behavior and his productions for light on two closely related questions, "To what anxiety is the patient's illness a kind of answer?" and, "By what means has the patient in the past met this anxiety problem?" Such information is extremely important for therapeutic strategy, for therapeutic efforts can then be intelligently aimed at finding the means whereby the patient can obtain the relief and gratification he needs without resorting to neurotic or psychotic reactions.

Why does not one always get this information simply by asking the patient what his anxieties are and how his illness affects them? One may get some interesting and valuable statements in this way, but one cannot rely on a neurotic or psychotic patient's direct answer to such a question to provide the necessary depth of understanding. So far as the patient's neurotic or psychotic forms of adjustment have alleviated his anxiety, to that extent he becomes unaware of it; consequently, the more neurotic or psychotic he is, the less he may realize that he has been solving his anxiety in that way, or even that he has had any anxiety. Furthermore, it is doubtful whether the patient is emotionally prepared to face clearly and appreciate fully the nature of the anxiety to which his symptoms constitute a defense; otherwise, he might not have needed to ultilize that kind of an escape or defense. He will probably need to gain some emotional support from the physician before he can face it. This is another illustration of the principle that personality study is inextricably interwoven with therapy.

Through considerable experience psychiatrists have come to appreciate and respect the general fixity and persistence of personality trends and the necessity of recognizing them and taking them into account in management and therapy. The expression of these trends becomes extreme in psychotic and neurotic reactions; this extreme expression may be moderated, but the underlying trend is not likely to be extirpated from the organization of the personality. This persistence of personality trends can be considered fortunate for society in some ways, for such trends in mildly exaggerated form may constitute the foundation for personal careers of great social value. Mediocrity is not, after all, the ideal norm of personality. Society has, for example, much need for persons of obsessive trend, provided it is constructively applied.

Sometimes the system of checks and balances by which a person has maintained a fairly consistent character in relation to others may undergo what seems to be a revolution, with the expression of new and different attitudes and traits. Organic disease of the brain may precipitate such an alteration of personality. A mood disorder may also do it. Such a chance is most noticeable when it is disagreeable to others. There may thus appear a meaner or more profligate aspect of the personality, which had previously been kept in check. To one who is interested in the intricacies of human nature, there is a certain fascination of tracing back the hitherto undercover existence of such trends and in working out the manner in which the previous balance had been maintained; but this is a task of considerable delicacy, which can at times create more trouble than it does good.

The commencement of training in personality study with patients in psychotic states offers an advantage in that such patients may exhibit these personality trends in such an excessive, caricatured fashion as to be obvious. A corresponding disadvantage may arise if the student comes then to recognize personality trends only when they occur in extreme form, for the chief value to the medical man of an understanding of personality trends is the insight afforded thereby into the adaptive limitations and possibilities (for good or ill) of all sorts and conditions of people who might become patients.

SENTIMENTS

For the gaining of insight into the personality trends of the patient and their relationship to his interpersonal issues, a fruitful field of study lies in a consideration of his sentiments or prejudices, that is, his attitudes toward father, mother, siblings and other significant persons, toward church and state, toward his home town and toward secret societies, anti-Semitism, socialism, fascism and other isms. In the discussion of such matters, the patient reveals more clearly than in response to direct questions the character of his ideals and the way in which he has come to dramatize his role in life. Probably the most significant revelations are in the expression of his wit and humor, rather than in the more formal verbalization of his sentiments. In an attempt at reconstruction of the development of the patient's sentiments, his earliest years repay close study. It may be said that the sentiments of the adult are the sediments of his childhood.

In studying the patient's past history and his own statements for information about his sentiments and prejudices, the physician should not neglect the information gained in nurses' notes and in casual observation of the patient's social behavior in the hospital situation. In dealings with hospital patients there is a tendency to look on all behavior as merely "symptomatic" of this, that or the other type of disease and, therefore, to neglect its personal significance. Helpful insight into the patient's attitudes can be gained from the study of the manner in which he establishes relationships with other patients and with the hospital personnel.

The patient's dramatization of the hospital situation often recapitulates the family situation of childhood. Two examples may be cited.

A young woman used to lie rigid in bed, yelling or muttering the same phrase over and over: "You people, we people; you people, we people." This went on day after day, with almost unvarying regularity, for about two weeks. As she grew more mobile, further developments and remarks made it plain that she was acting out in a confused and exaggerated way the tensions and antagonisms of her childhood home. She had an Irish Catholic father and a mother of old New England stock—"we people; you people."

A manic woman aged 21 used to make fun of her doctor, who was rather stout, dignified and patronizingly tolerant. She teased him about sex and religion. When he came to the ward, she would yell out, "Here comes the —— bishop. He's got a 'hard' on today." She had been a rebellious, delinquent daughter of a rigidly moralistic missionary, who had fathered fourteen children.

NEED FOR FORMULATION OF PERSONALITY PROBLEMS AND TRENDS

All of the sources of information which have been under consideration yield data for the case record, but the material needs to be formulated in order to yield a useful insight into the patient's inner conflicts and their possible solution or alleviation. If the patient remains under continued treatment and study, it is well to reformulate the patient's situation and problem from time to time, after more knowledge has been gained and some of the inevitably false information has been straightened out.

Repeated interviews bring decided advantages in personality study. A period of reflection should be systematically scheduled after every interview. The subsequent interview then gives one a chance to confirm or disprove or modify one's tentative formulations. A day's pause may also bring about revealing changes in the person studied. He may be found in a different mood. His "second thoughts" may bring out more significant information. He may have gained some needed self respect or courage from the first interview, and with that support he may open up on matters previously concealed or "forgotten."

The data of the medical history, the physical and neurologic examinations and the special psychometric and laboratory tests must also be evaluated in order to arrive at a sound formulation.

In the following section, additional suggestions are offered for recognition and formulation of problems and trends concerned with the personality. These suggestions are offered not as a system of explanations but as conceptual aids to the interviewer in getting a grasp of the biographic and descriptive facts and in organizing them into an understandable pattern. When

this has been well done, the facts will tell their own story, with a minimum of psychiatric "patter."

RELATION OF PERSONALITY TRENDS TO INTERPERSONAL ISSUES

When the material concerned with the patient's conflicts and personality trends is reviewed, it will frequently be noted that it has a close relation to certain interpersonal issues which can be discerned in the data on the patient's personal history and social adjustment. It can be stated, as a first approximation to the truth, that a person's personal conflicts arise from interpersonal issues between himself and others and that his outstanding personality trend represents the habitual way in which he has come to resolve some interpersonal issue.

For example, some of the issues, conflicts and trends concerned with the need for affection may be considered. Affection from the parents, or some equivalent thereof which leads to the care of the young, is a basic biologic feature of life in the higher mammals. The need for the parent's affection is the young child's most fundamental incentive to domestication and social conformity. In later childhood, another important incentive to social conformity comes from the desire for the respect of his fellows. Opposing these incentives to conformity is a strong human desire for independence and resentment of control, which reaches a peak in adolescence. A variety of interpersonal issues and personal conflicts may develop out of these contrary desires. When the affectionate response of parents, or other loved persons, is felt to be inadequate, insecurity and anxiety result. Almost every one reacts at some time to such apparent neglect by behavior which gains attention, even though it has to be misbehavior. Such attention-seeking tendencies are a common means of fulfilling a definite childhood need; yet they commonly evoke punishment or threats. When punishment leads to resentment, there is a conflict between the resentment and the desire for affection. Some persons succeed, in the main, in stabilizing this conflict by an attitude of extreme filial devotion, which serves, rather automatically, to assure affection and security; but such a solution may be hindered or blocked by resentment.

The frustrations of some of the child's natural propensities during the processes of domestication and socialization may also set up conflicts. In general, the desire for the feeling of security afforded by affectionate relationships more than balances these antagonistic impulses. It is important to recognize, however, that the usual course of life regularly leads to the development of certain areas of conflict. The fact that such conflicts do not remain sharply in the foreground of conscious attention does not mean that the conflicting feelings and impulses have no meaning for the behavior. In a fashion somewhat analogous to that which somehow maintains a "conditioned reflex," the organism maintains unconsciously many patterns of meaning, focused on certain significant persons, and these complexes of meaning frequently include conflicting tendencies. Personality trends develop as systematic ways of dealing with the anxiety generated by such conflicts.

Obsessive Trend.—Resentful and aggressive impulses against persons on whom one is dependent for security form a prolific source of anxiety, experienced as a sense of guilt. Substitutive types of "goodness" may be developed, their degree of compulsion roughly approximating the degree of guilty anxiety arising from the unacknowledged resentment. Uncalled-for gestures of appeasement or perfectionistic rituals are the more obvious types of "making up for" such guilt or anxiety. This is the line of response which is developed into the obsessive trend. Such an obsessive trend may include excessive orderliness; meticulousness; exaggerated need for cleanliness, precision or pedantry; perfectionistic strivings; perseverative tendencies; ruminations; a habit of harping on subjects; hair-splitting argumentativeness; adherence to time-table routine in living, and extreme dependence on rituals. In the psychoanalytic school of thought, such obsessive developments are presumed to be derived from anal erotism and the formation of reactions thereto. Persons who show such obsessions are then called "anal characters." These statements are not so fantastic as they may appear at first blush. The housebreaking of infants is an early and important issue for the interaction with parental praise or punishment to which the obsessive trend may be one person's type of response.

Even with a well developed trend in the obsessive direction, certain persons may find such a solution inadequate to resolve their conflicts and then may suffer from anxiety attacks, or the undercurrent of resentment may break out spasmodically into the overt expression of defiance (the episodic expression of a self-assertive trend).

Hypochondriacal Trend.—Other persons may be inclined to solicit the much desired affection through illness and so, quite without planning or intending it, may come to behave automatically in ways which might gain sympathy. Periods of convalescence from the illnesses of childhood provide early opportunities for the cultivation of

this tendency. Sympathy, like attention, is an almost satisfactory substitute for affection. A well established habit in this line shows itself most characteristically in the automatic magnification of trifling pain or distress and a great interest in the body functions. This is called the hypochondriacal trend. This trend may be greatly strengthened by an oversolicitous mother, especially when the mother's oversolicitousness represents an obsessive trend to make up for some hostility within her.

For the person starting to cultivate a hypochondriacal trend, a fertile source of further preoccupation with bodily complaints lies in the physiologic expressions of his anxiety, such as unsteadiness of the heart rate, pronounced motor or secretory activity or inactivity of other viscera, aches in the head, neck or back resulting from the muscular tension of the anxious state, or the conversion of mild itching into severe excoriations by frequent scratching.

Neurasthenic Trend.—A closely related trend, which also often shows itself in neurotic illnesses, is the tendency to anticipate frustration or failure by magnifying the difficulties which would excuse failure. If a person has had to gain acceptance by performance, that is, if he has not been able to feel that he is accepted with affection, on a personal basis, then any lack of success or the expectation of falling short of what would be considered worth while may be so great a threat to his security as to generate excessive anxiety, unless some excuse, like fatigue, can be manifested. Persons who are driven by a sense of duty rather than by enthusiasm are especially prone to magnify fatigue. The clinical picture called neurasthenia is so common a manifestation of this trend that it may be spoken of as the neurasthenic trend. Neurasthenic trends often accompany obsessive trends, serving to relieve somewhat the excessive sense of duty.

Not infrequently, in investigation of the family background of persons with obsessive trends who work themselves into neurasthenic states, it will be found that there is a close relative rather strikingly deficient in a sense of duty, for whom the patient is unconsciously striving to set an example of "determination."

"Constitutional Inadequacy."—There is a large number of persons with vague and ill defined distresses and pains who seek vainly for relief, going from doctor to doctor and from one ill advised operation to another. A good proportion of them would be much better off if they could acknowledge their limited endurance and live within their means, physiologically speaking. Alvarez has proposed the term "constitutional inadequacy," which is probably a useful description so far as it emphasizes the generalized nature of the disorder and the improbability of explaining it by reference to a specific organ and the impossibility of curing it by a specific ectomy or specific medication. The constitutional designation, so far as it implies a hereditary cause, is somewhat too sweeping in its neglect of the personal, experientially determined, aspects and the possibility, in some instances, of much relief and increased effectiveness through emotional reeducation. Some of these patients have obviously poor physical endowment; others may have less obvious physiologic limitations, but many, in addition, are effort-addicted persons who do not feel "right" unless they are pushing themselves beyond their margin of safety. Constrained by this obsessive trend, they sentence themselves to a miserable alternation between neurasthenic invalidism and what is for them obsessive overwork. At the times when they appear in hospitals or clinics, they customarily present a neurasthenic picture with hypochondriacal features, but the obsessive trend constitutes the major difficulty in psychiatric treatment.

Addiction.—An escape from anxiety and guilt is made possible momentarily by the use of alcohol, ether, opiates, barbiturates, cocaine, cannabis or other narcotic substances. Indulgence in such a method of escape through partial narcosis may become a firmly established reaction pattern, comparable to other neurotic habits of evasion and requiring similar study and treatment.

Paranoid Trend.—A decidedly different solution of the guilty anxiety over certain issues is found in the paranoid trend—the tendency to attribute hostile or aggressive motives to others. The "content" of a paranoid trend—the particular motives and intentions attributed to others—may mirror the patient's own inclinations. Some persons have not achieved an adequate perspective about themselves. They are lacking in insight and humor and cannot endure to admit the existence in themselves of some of the "baser" inclinations and propensities of human nature. If, for example, in the company of another person they become aware of improper sexual feelings—especially "perverted" or "inverted" inclinations—they may attribute this experience to a "base influence" of the other person, or even deny their own emotional responsiveness and try to dispose of the whole matter by attributing "designs" to the other person. In the degree to which it is successful the paranoid trend relieves the patient's own anxiety over guilt by his blaming the other fellow. The reaction is somewhat analogous to the political device by which a nation may allay domestic

discord and avoid civil war by a "defensive" foreign war.

Persons who have felt a great need for affection, and who are therefore sensitive when others do not live up to their heavy demand in this regard, are particularly prone to feel slighted and readily acquire paranoid tendencies.

Other trends influence the paranoid trend. The hypochondriacal trend—preoccupation with symptoms and signs of illness in oneself—may supply the patient with ideas of being poisoned or otherwise injured in body by the persecutor. An obsessive trend of the perfectionistic type, when combined with the paranoid trend, may incline him to suspect that the persecutors are accusing him of wrongdoing—"They are spreading lies about me."

The paranoid tendency to put the blame on others has a parallel in the tendency to self magnification. In psychotic states the combined tendency is often expressed in combined delusions of persecution and of grandeur.

Tendency to Daydreaming and Delusional and Hallucinatory Preoccupations. — In a mildly pathologic way, consolation for unsatisfactory relationships in real life may be found in daydreaming and in self-flattering delusions. In a healthy and constructive way, some kinds of imaginative playing with mere possibilities may be a useful and valuable offset to the obsessively realistic preoccupation with the actual. Scientific discoveries, inventions and social vision come about through dissatisfaction with things as they are and the use of creative imagination as to how things might be different. Such trends, however, may take an unhealthy turn. When the sense of responsibility is slight, or the incentive to conformity is weakened, there may be little to recall one to that regular habit of comparison with the consensus of the group which constitutes "reality" in the practical sense; and one may then remain seriously disabled for practical life through a delusional and hallucinatory preoccupation with nonpractical issues. It is surprising that only a few people are grossly and persistently deluded, for human beings quite regularly react, without much logical reflection, in ways determined by habit, sentiment and momentary desire, and then tend to rationalize their behavior by believing that which justifies such reaction. The fact that gross delusions are uncommon is attributed, in large part, to the strong human tendency to conform to generally accepted premises and obligations and, in part, to the capacity for plausible juggling of accepted premises into line with behavior which may be some distance out of line.

The tendency toward delusional consolations does not ordinarily succeed promptly in gaining a gratifying delusional escape from "reality." Internal conflict and internal debate, with hallucinatory representations of the issues involved, may be prolonged and relatively unpleasant. Not every hallucinatory experience is by any means a direct wish fulfilment; many hallucinations are insulting and antagonistic, representing "the other side" of a personal issue.

These varied tendencies toward fantastic daydreaming, hallucinatory preoccupations and beliefs which are at variance with the practical consensus of one's fellows may be usefully designated by the term delusional trend. The question whether, in a given person, such a delusional trend comes into prominent expression is determined not so much by the special force of this tendency as by the failure in those integrative forces which hold the person in order.

In delirious and confusional states resulting from infection or exhaustion delusional trends often appear. In the absence of confusion, pronounced delusional manifestations may ordinarily be taken to mean that there is a serious distortion of the personality, as with schizophrenic disorders. The delusional trend is, therefore, scarcely to be considered as a primary force in development of personality. It is, rather, a secondary influence, the clinical importance of which consists in shaping the clinical picture of a disorganized person. The glimpses into the patient's personal preoccupations vouchsafed by delusional statements may, however, have great importance, as do dreams and other fantasy productions, in revealing to the physician significant aspects of the patient's personal problems.

Dissociative Trend.—Adolescent conflicts about sex are sometimes "solved" by the extraordinary dodge of losing a function. One may lose one's voice or one's hearing or sight; one may become paralyzed in the left leg or anesthetic on the right arm up to the edge of the sleeve, or one may "forget" certain matters. The falling away or separation of such a particular ability from the control or use of the patient without some organic lesion to explain it is attributed to a mental reaction called dissociation. This is the essential feature of what in modern times is called hysteria. The tendency to resolve conflicts by resorting unconsciously to such losses may be called a dissociative trend. When, for example, one feels sex as degrading or disgusting, yet is thrilled and incited toward mating behavior, a possible mode of defense against such an anxiety-producing conflict lies in the rejection of sensation in those parts whence thrills have come or in the rejection of power over those members

the activity of which brought thrills. When avenues of incoming erotic stimulation or of outgoing adventurous search for such stimulation are blocked, one is immured in an ivory cage of "purity" and yet gets a derivative ascetic or saintly gratification. This trend, being in large measure a reflection of current ideas of "purity," is subject to wide changes in fashion and subtle transformations.

Sex is not the only source of shame which can serve as incentive to hysterical dissociation. Sensory and paralytic disabilities brought about by dissociation have been observed in military life, as automatic mechanisms for avoidance of what might otherwise be a dreadful disgrace. Memory dissociation, in the form of "fugue" or "amnesia," has also provided escape, in some instances from financial disgrace.

Organ Neuroses and "Psychosomatic" Conditions.—In these disorders a particular reaction of an organ or a system has become for the individual patient the expressive outlet of a particular personality conflict. Colitis, asthma or dermatitis, for example, may become involved in such a personality reaction. Early experiences of physical illness at times of interpersonal conflict may "condition" these patterns of organic reaction so that they serve as the regular response to later similar conflicts.

Repetitive patterns of disturbance of the activities of organs and tissues concerned in anxiety reactions may be important influences in the pathogenesis of indisputable organic disease, such as peptic ulcer or coronary sclerosis, or other conditions, such as hypertension, hyperthyroidism, cardiac decompensation or arthritis.

The "Don't Mind" Trend.—One of the most seriously disabling trends is difficult to put into words. One habit of self defense against the anxiety over lack of affection may be expressed in the words, "Nobody cares for me; well, I don't mind; I don't care for anybody else." When actually expressed in words, this reaction may not be so disastrous as when it is kept at a deeper level. With the more outspoken expression of resentments, such a personality development may be in the direction of trouble-making delinquency. With greater success in "not caring," the patient may make a schizophrenic adjustment to life. "Don't mind," translated into Latin, suggests "dementia," and "don't mind," translated into life, also comes out as something very like "dementia."

Constructive Trends and the Integrative Forces of Personality.—The preceding discussion of personality trends commonly encountered in clinical work leans heavily toward the pathologic. But picking a man to pieces in search of abnormal trends is an inadequate sort of personality study. The purpose of interviewing, and of the clinical study of personality, is a constructive and integrated grasp of the person as a going concern—not merely a delineation of the pathologic features, but an appreciation and evaluation of the assets and the constructive uses of even second rate patterns of reaction. To speak figuratively, as well as literally, limping may be "caused," so to speak, by the deficiency of certain muscles the innervation of which has been destroyed, for instance, by poliomyelitis; but it is not the injured muscle which limps—it is the man, managing somehow to keep going despite the defect. Study should be aimed at finding, and therapy, at helping him use, whatever means may aid the man to do better.

Perspective on Personality, Situation and Reaction.—In a recapitulation of these suggestions for interview and personality study, a certain progression will be noted. Starting with a consideration of the presenting problem, the examiner notes primarily the patient's attitude toward this problem and the persons and responsibilities involved in the situation. Next, he gathers evidence about the conflicts which generate anxiety in the patient and about the personality trends by which the patient has come to react, customarily or episodically, to his particular conflicts. Then, the examiner seeks information about the interpersonal issues in life about which these personality trends have developed.

The lengths to which such a study should be pushed in the individual case depends on a number of considerations. As was stated earlier in this guide, some patients may manage very well to establish more order, peace and security in their personal life with only simple enlightenment. Much depends, also, on the understanding and personal qualities of persons who are nearest the patient in the home situation.

In cases of great complexity the patient may need a great deal of help in getting a better perspective on the three main features of his problem: (1) his personality, (2) his reactions and (3) the situation to which these reactions occur.

There is often much confusion about these features. Commonly, the patient who is reacting with the physiologic expressions of anxiety thinks that these constitute his illness and does not understand that the essential difficulty arises from some incompatibility of his attitudes or from habitual reaction patterns which render him ineffective in settling certain personal issues. Perhaps the greatest difficulties arise in the misunderstanding of situations. Life situations are not adequately defined by the circumstances. Circumstances are merely the raw materials out

of which people shape up situations, automatically, in terms of their personality organization. The issues are shaped by attitudes as much as by so-called facts. Nor does the physician understand adequately the theme of the patient's life situation until he recognizes and evaluates the attitudes which determine the issues involved.

DEFINITIONS AND TERMS RELATED TO PERSONALITY

SOCIAL DEFINITION OF PERSONALITY

Up to this point no definition of personality has been offered. There is, of course, much greater practical value in knowing how to deal with personality in action than there is in expressing the meaning of it in words. The usual definitions are somewhat too broad to be useful. Warren and Carmichael [4] stated: "Personality is the entire organization of a human being at any stage of his development." Menninger [5] defined it thus:

> Personality is the individual as a whole, his height and weight and loves and hates and blood pressure and reflexes; his smiles and hopes and bowed legs and enlarged tonsils. It means all that anyone is and all that he is trying to become.

The narrowness of the following definition (its restriction to the social significance of personality) may have a certain advantage of tangibility and definiteness: Personality is the organized system of sentiments or attitudes by which one establishes relationships with others and negotiates interpersonal transactions.

In further elaboration of this definition, it may be added that one's sentiments are one's modes of emotional response to and expectation from others, as developed and fixed through interpersonal experiences; that the particular sentiments already established at a given stage of one's personal history set the patterns by which other persons then are accepted into significantly personal relationship, and that sentiments are relatively resistant to reasoning but are somewhat modifiable even late in life, through emotional experiences in personal relationships. The quick appeal which one's personality has for others is largely determined by the range, flexibility and vivacity of one's sentiments. (The man in the street calls this "lots of personality.") One's more enduring personal value to others is largely determined by the stability and integrity of one's sentiments. (The man in the street calls this "character.")

4. Warren, H. C., and Carmichael, L.: Elements of Human Psychology, Boston, Houghton Mifflin Company, 1930.

5. Menninger, K. A.: The Human Mind, New York, Alfred A. Knopf, 1937.

TEMPERAMENT

It is the possibility of discrimination of temperamental characteristics in a relatively independent fashion that permits the definition of personality in as narrow a social sense as has been done here. The term "temperament" refers to certain emotional qualities and characteristics which limit or condition the manner and effectiveness of functioning of the personality, whatever the issues and trends. One of the most important of such considerations concerns the energy management of the organism—whether a person is geared, as it were, for steady and fairly uniform effort, with steady rhythms of application and rest, or whether he is fitful in energy control, perhaps capable of brief effort at high pitch, with quick fatigue.

INTELLIGENCE

Another relatively independent variable which commonly needs to be evaluated in personality study is the capacity called intelligence. Intelligence may be defined as the level of innately limited capacity for modification of behavior through experience and trial. A fully adequate test of the limits of the constitutional endowment of intelligence is probably never attained. Under comparable conditions, people acquire knowledge and mental skills in some rough proportion to their intelligence; hence, such acquisitions give useful clues to its estimation. Special standardized tests are often used for this purpose, such as the Stanford revision of the Binet-Simon test and the Wechsler-Bellevue tests.

"CONSTITUTION," HEREDITY AND ENVIRONMENT

In the psychiatric study of a patient, it is customary to give special attention to his "constitution." In one of the senses commonly implied, the "constitution" is something determined by the chromosomes—fixed at birth, unalterable and determining the person's behavior through the vicissitudes of life, like the inexorable finger of fate. There are rare organic diseases of the nervous system which seem to be constitutionally determined in this sense. It should, of course, be recognized that chromosomal mechanisms do not operate in such ways as to produce offspring exactly like the parents.

In a looser sense, "constitution" is a term used to cover the more persistent personality characteristics, with two subsidiary implications: first, that the person's constitution is closely similar to that of others in the family and, second, that the personal family traits are carried along in some fashion by inheritable characteristics of

the organic structures of the body. Because of these implications, a certain amount of study has been given to the correlation of body type with personality characteristics and disorders. The statistical data justify the statement that there is some degree of correlation—enough to stimulate further study but not enough for diagnostic reliability.

Attempts to discriminate between the effects of chromosomal heredity and social influences are usually blocked in the individual case study by the fact that the very relatives through whom hereditary characteristics might be traced are usually the most significant figures, also, in the environment, so that their own personality trends and personal conflicts permeate the social milieu of the offspring. For example, as was mentioned earlier, there is a tendency for obsessively overprotective mothers to influence their offspring in the hypochondriacal direction. Great difficulty is encountered in attempting clinically to evaluate separately the social mechanisms of inheritance and the chromosomal mechanisms. There is, however, a good deal of practical value in formulating the available information, sketchy though it may have to be, concerning the personalities of close relatives and household associates of the patient.

There is also a great deal of practical value in the physician's adjusting his therapeutic aims in the individual case by a realistic appraisal of the "constitutional" limitations of the patient. There is, however, much need for more sound and specific knowledge in this field. As in other bankruptcy situations, one needs to be on the lookout for concealed assets and unsuspected advantages.

ADJECTIVES AND EPITHETS ABOUT PERSONALITY

Introverted or extroverted; schizoid or syntonic; altruistic or selfish; sanguine, melancholic, choleric or phlegmatic; volitional, mental or vital; outgoing or shut-in; aggressive or submissive—such a check list as this may be employed for the hurried summarization of personality.

This "name-calling" system of personality study is not recommended in this guide, because the functioning of personality represents so complicated a system of interacting dynamic forces that "over-all" epithets seldom fit well.

This epithetic method of personality description can be made more useful (1) by stating the persons, or groups of persons, toward whom the subject shows the characteristics described and (2) by indicating the typical situation or mood in which his behavior may be so characterized. On the whole, however, a more useful characterization of a given personality is provided by a delineation of his personality trends and the issues and themes which are discernible in the critical events of his life.

STAGES OF EMOTIONAL GROWTH

MATURITY, OR THE STAGE OF EMOTIONAL GROWTH

An important part of the personality study consists in evaluation of the patient's emotional maturity. How does one clinically judge a person's maturity? It is a task similar to the evaluation of insight. Insight, as practically judged, concerns the perspective in which the patient sees his presenting problem. The level of maturity, likewise, is revealed by the perspective in which he sees his life and work. Just as there is no one absolutely correct insight into, but, rather, a variety of reasonable formulations of, any presenting problem, so there is no single pattern of adjustment which could be called "maturity."

In his studies on psychosexual development, Freud disclosed one of the most valid and sensitive indicators of emotional maturity in adult patients—the capacity for a mutually and thoroughly gratifying erotic relationship with a mature mate. This is just another example of the principle that the facts regarding one's psychosexual development reveal with exquisite fidelity the character of one's past interpersonal issues and general personality trends, probably because the sexual relationship is so definitely interpersonal and forms such an intimate and pervasive part of life. In clinical work, such a valid knowledge of details about a person's intimate life as to make this psychosexual criterion of maturity practically useful is not likely to be available until confidential rapport has been reached, which means, in effect, that it turns out to be most useful after the physician has gotten some distance in a treatment relationship.

Perhaps most psychiatrists, being middle aged, would regard as "emotional maturity" the set of attitudes characterizing middle age, thereby implying some superiority for attitudes of caution, tolerance and easy-going acceptance of things as they are, in contrast to the impetuous urgency and competitive strenuosity of earlier years. It is more useful, however, to eliminate the idea of 100 per cent maturity in human development. Human nature is not like canteloupe or peach, with just one perfect moment of absolute maturity. Rather, there are successive stages of development in interpersonal attitudes which characterize, roughly, different age groups, and a person's maturity or immaturity

must be expressed as relative to these stages. To state or to imply that Johnny Jones, aged 18, is immature is not very helpful. It is more helpful to state specifically to which earlier stage of development his attitudes belong. Although there is no generally accepted scale of emotional maturity comparable to the well established scales for intellectual development, the following rough characterization of several stages of emotional development may be useful:

Infantile Stage.—This stage is characterized by a complacent dependence and the expectation of infinite service and infinite tolerance from others, with petulance when the wishes are not immediately gratified. (There are some persons, chronologically middle aged, who are emotionally almost infantile in this sense.)

Childish Stage.—This stage is characterized by a limited range of secondary responsibility, with the expectation that this "responsibility" will be canceled by the all-powerful parent by reason of any good excuse or ignorance. There remains, however, at this childish stage, a need for complete trust in another as the basis of any acceptable relationship. (The usual patient, when physically very ill, temporarily regresses to this childish level, and his need for complete trust in some one is a serious and important fact in medical management. Duing convalescence the patient usually becomes again less trustful and more self assertive.)

Early Adolescent Stage.—This stage is characterized by much concern over independence from parents and by hero worship (that is, admiration for and devotion to extrafamilial figures, frequently subject to explosive disruption), as well as by much interest in gangs, badges and exhibitionistic rivalries, with everything pushed to extremes, and by a self-conscious awareness of sex.

Late Adolescent Stage.—This stage is characterized by a pose of "sophistication," a "sophomoric" attitude or a "line" of chatter, witticism and wisecracks, whereby an appearance of greater maturity is sought. There is a tendency toward extreme idealism or cynicism or an idealization of aggressively strenuous competition, with much preoccupation with personal independence.

American culture sets a high value on the "independence" and strenuosity characteristic of this stage and so tends to perpetuate the late adolescent style of emotional attitudes into adult years; nevertheless, most Americans, as they advance into the twenties and thirties, become comfortably reconciled to the practical realities of interdependence in economic and professional matters, lose some of the emotional urgency of competitive striving and accept candidly their own limitations and the limitations of others without feeling necessarily defeated thereby.

It is not an easily described clinical task to evaluate a patient's stage of emotional development. In ordinary clinical work, however, this task is pointed up in a practical way by the evaluation of maturity with respect to the outstanding feature of the patient's life situation, which is brought out by the theme of his main preoccupation.

Unevenness of Personality Growth.—In the characterization of a person's stage of emotional growth, it is well to recognize that he may not progress equally in all respects or relationships. For example, a man may be more mature in his professional and business life than with his wife, or a woman may be more mature in her home life than in church or club activities, and vice versa. One may, under certain conditions, revert to immature attitudes in certain respects and yet hold to a more mature attitude in others.

THERAPEUTIC IMPORTANCE OF THE STAGE OF EMOTIONAL GROWTH

An appreciation of the patient's stage of emotional growth has considerable therapeutic importance. Observations on relatively trivial items, such as the gait, the patient's posture or tone of voice, or his manner of making requests or of expressing appreciation may when viewed as indications of the stage of development of the personality contribute appreciably to a sympathetic insight into the nature of his deeper problems, provided the interviewer has some knowledge of the usual steps in the growth of personality and an appreciation of the interpersonal problems which are characteristically important at different stages. By a well founded evaluation of the patient's stage of emotional development, the physician may avoid futile and confusing efforts to use incentives that have no constructive meaning at that stage. Furthermore, such insight provides helpful hints of possible interests and activities natural to the patient's next stage of personality growth. Perhaps the most direct and immediate therapeutic value of such insight lies in its effect on the physician's attitude. It enables him to see through the surface marks of an apparently higher stage of development and, hence, to avoid the irritability and exasperation engendered by the failure of the patient to meet expectations which are inappropriate to his actual stage of development.

The psychotherapeutic strategy in cases of serious personality disorders is concerned largely with the problems of the patient's further growth

in personality, beyond the frustrated stage of his illness. The interaction of the patient's attitudes with those of the therapeutist and his associates comprises the main tactical measure. Respect for the individual personality and a considerate and appreciative understanding encourage the patient's spontaneous activity and his assumption of a responsible and gratifying role. With such help the patient may resolve some "unfinished business" which may have obstructed his emotional growth.

ORGANIZATION OF A PSYCHIATRIC CASE REPORT

In the presentation of a case study for psychiatric conference and discussion, it should be remembered that interpretations and formulations do not arise spontaneously from the facts. They represent thought and judgment, either frankly and intentionally expressed or concealed as descriptive prejudice. Within tolerable limits, one's fellow workers appreciate a brief consideration of alternative interpretations and judgments, but not to the point of confusion or to the neglect of responsible decision.

The following order of presentation is suggested as that best designed to enable the hearer to grasp the significance of the facts:

1. Give the identifying data: name (when permissible), age, sex, marital condition, occupation and socioeconomic status, with some outstanding feature which may help the other members of the group to recall their acquaintance with the case.

2. State briefly the presenting problem (or chief complaint) and the patient's attitude toward it.

3. Tell how the presenting problem developed (present illness).

4. Describe the patient's general behavior and attitudes and the simpler tests of his capacities, including portions of the actual interviews.

5. Characterize briefly the current personality reaction pattern in common clinical descriptive terms (personal diagnostic formulation, paragraph 9 A).

6. Present the pertinent information from the medical history, the physical and neurologic examination and the special tests. If the evidence justifies the diagnosis of an organic disease of the brain, a toxic condition or a troublesome somatic disorder, say so at this point and indicate how it limits or hinders the personality functions.

7. Give an integrated discussion of the personality issues and trends involved in the presenting problem, the present illness, the past personal history and the family history, that is, a biography focused on the personality and its development, with special reference to the current reaction type.

8. Select a diagnostic label for statistical purposes, with a brief consideration of reasonable alternatives.

9. Sum up the significant facts and your judgments about them in a brief personal diagnostic formulation, indicating the aim and the general plan of treatment.

A. The clinical picture, or pattern, of the present personal reaction.

(*a*) Adjectival characterization: A brief characterization by such terms as delirious, confused, amnesic, panicky, hypomanic, manic, agitated-depressive, retarded-depressive, anxious, neurasthenic, obsessive, catatonic, hebephrenic, paranoid, hypochondriacal, dissociative, asocial, self blaming or self praising, or combinations thereof.

Destructive trends, whether suicidal, homicidal, assaultive, mutilative or operation seeking, should be especially noted and evaluated and underlined.

(*b*) Organization of Sentiments now Dominant: In what patterns of personal relationship does the patient now bring about, or permit, personal contact and relationship with others?

B. Biographically established characterization of the patient's constitutional and temperamental assets and liabilities, personality trends and special emotional needs and vulnerabilities, as developed in the familial setting and exhibited in prepsychotic life. This should be compared with the organization of sentiments now dominant and with the patient's ideal of self. Occupational success, interests and social values and codes are considered.

C. Analysis of the current personal issue and the life situation.

(*a*) The "complaint problem" and the patient's attitudes toward it and toward the life situation and the responsibilities involved.

(*b*) The potential advantages and disadvantages in the present life situation for the satisfaction of the patient's emotional needs by the type of reaction shown.

D. Alternative possibilities by which this particular patient may resolve this situation more satisfactorily for his needs, with due consideration for the welfare of others; probabilities.

E. Immediate working plans.

SUMMARY

The interview, as the primary technical psychiatric procedure, is a part of the medical examination of a patient—the part concerned

with elicitation of the patient's attitudes and his patterns of interpersonal reaction, through the study of his reactions in the interview and in his biography. For clinical working purposes personality may be defined as "the organized system of sentiments or attitudes by which one establishes relationships with others and negotiates interpersonal transactions." A number of specific personality trends are related to the interpersonal issues which may have been arousing anxiety.

The biographic account of the patient's development, infancy, childhood, adolescence and adult life, of different attitudes with regard to dependency and responsibility, and of the varying methods typically used at these periods for assurance of affection and respect, provides the physician with useful suggestions for evaluating the patient's emotional maturity or stage of emotional growth and for utilizing such observations and formulations in planning psychotherapeutic strategy to help the patient in finding more suitable and more gratifying attitudes and reaction patterns for meeting the situations which arouse distressing anxiety. These considerations have medical significance because such anxiety may persistently disturb physiologic functions or because an automatic resort to some neurotic or psychotic pattern of defense may leave the personality "sick," i. e. crippled or incapacitated.

Henry Phipps Psychiatric Clinic.

NEURONAL DISEASE ASSOCIATED WITH INTRACYTOPLASMIC INCLUSION BODIES

JAMES W. PAPEZ, M.D.
ITHACA, N. Y.

Striking alterations in neurons are often seen in the brains of patients who had manifested various clinical symptoms. There appears to be present a primary neuronal condition, which begins with hyperchromatic alterations of the Nissl material, progresses to a ragged, tattered appearance of the nerve cells and leads gradually to alteration of the chromatic particles, formation of vacuoles in the nerve cell and deformation of the nerve cell and its dendritic processes. When this stage is reached, the disease can readily be recognized under the microscope. Then follows a stage of impoverishment of the nerve cell and degeneration of its cytoplasmic constitution, leading to formation of a shadow cell; the process may pass on to final dissolution, represented by fragmentation of the structural protein of the cell body and the remnant of a naked nucleus. When the process is slow and inconclusive, local areas in the cell may show encrustations of pigment and other signs of structural degeneration.

In different cases various stages of this neuronal disease are represented, and its presence is suspected only when the degeneration of the neurons has advanced to the stage of deformation and vacuolation of the cytoplasm, and sometimes to pigment formation.

In the past two years these changes have been repeatedly observed in cases of a number of conditions, including spastic athetoid paralysis, juvenile spastic paralysis, chronic chorea, postencephalitic paralysis agitans, pituitary basophilism (Cushing's disease) and disturbances recorded as terminating in psychoses. In 2 of these cases the disease had a sudden onset. In most of the others the onset was insidious, and in all progress was gradual. In 2 cases the illness terminated in a psychosis; in 1, with pneumonia following an operation on the brain, and in 1 case a patient with spastic paralysis lived to the age of 67. Without discussing here the clinical symptoms and the more general neuropathologic details, I shall present only the more striking neuronal changes, as seen microscopically in these cases.[1]

From the Department of Zoology, Cornell University.

The presence of a pathogenic agent was suspected from the appearance of the Nissl substance with ordinary stains. Various staining technics were tried. Finally, a method was found and perfected which brought into relief round intracytoplasmic bodies with characteristics that led to the conclusion that they are living protein bodies or particles capable of multiplying into colonies in the cytoplasm of the nerve cell, of living in symbiosis with the nerve cell for a long period and, when discharged from a disintegrated cell, of migrating in groups, and sometimes singly, in the nerve tissue. The nature of the round particles, or cytoplasmic inclusions, was not determined, but their characteristics and behavior were studied in a variety of pathologic stages. These inclusions were noted in small numbers and in a sequestered form in brains which were presumably normal. However, 3 brains examined showed none of these inclusions in the regions from which the sections were taken. Properly stained sections from a number of other brains failed to show these characteristic inclusion bodies.

NEURONAL PICTURE AS REVEALED BY ORDINARY STAINS

Treatment of material with the ordinary cresyl violet stain or with other methods of staining the nucleus, the cell body and its Nissl substance revealed the features of the disease to be described. The inclusion bodies as a rule were not rendered visible by the ordinary staining methods and might be completely obscured by the more obtrusive Nissl substance.

1. Colonel J. E. Ash, Medical Corps, United States Army, and Captain W. Haymaker, Medical Corps, of the Army Medical Museum, Washington, D. C., furnished material in the case of pituitary basophilism and in several other cases; Dr. W. B. Hamby, of Buffalo, material in the case of postencephalitic paralysis agitans; Dr. A. B. Sabin, of Cincinnati, the specimens of Toxoplasma infection in a mouse; Dr. A. R. Vonderahe, Cincinnati, the brain in a case of spastic hemiplegia and the brain in a case of early chorea followed by paraplegia in flexion, and Dr. H. W. Ferris, the brain of a child with spastic athetoid paralysis, from the Reconstruction Home, Ithaca, N. Y.

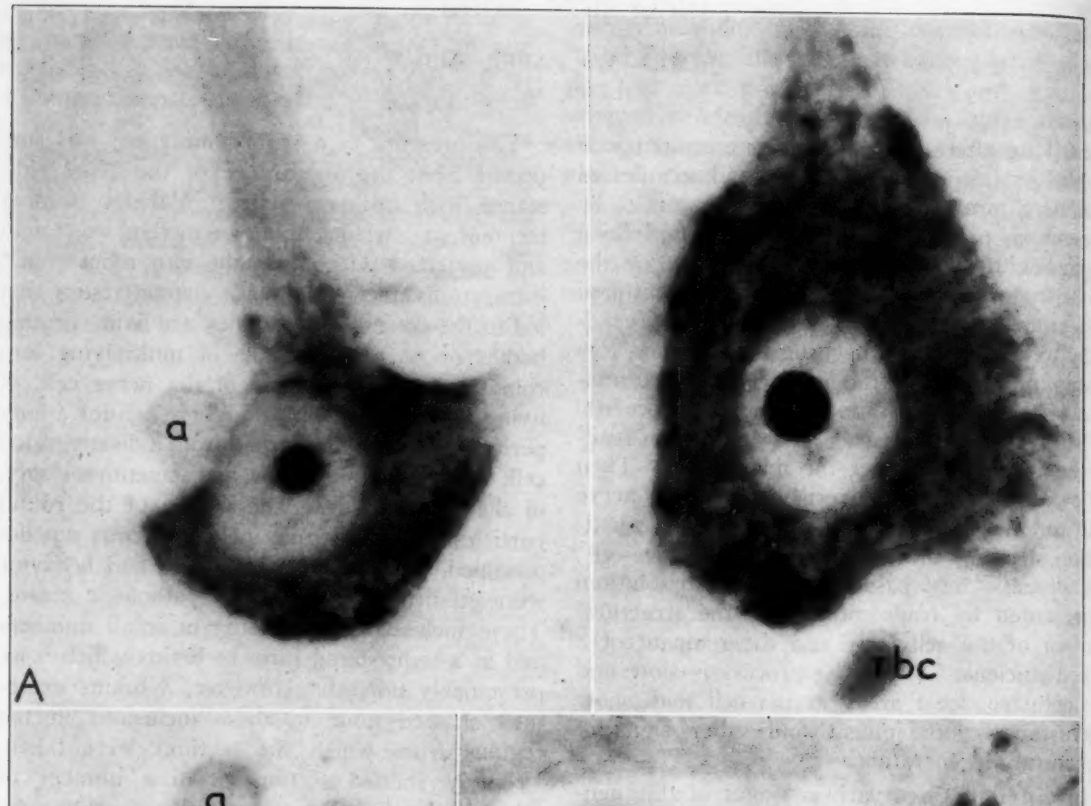


Fig. 1.—*A*, hyperchromatic neurons in a case of postencephalitic paralysis agitans, stained by the ordinary cresyl violet method. Around the nucleus is a granular mass (*i*) containing the inclusion bodies. *B*, a nerve cell containing only a granular mass (*i*) and another neuron with a ragged surface. At a certain stage the ragged surface is more prominent than is that in the cell shown. Nuclei of protoplasmic astrocytes appear at *a*. Magnification, 2,475 (objective, × 120; eye piece, × 10; bellows, × 2.06; slight reduction).

Six stages were identified, five of which seemed to represent a sequence of neuronal reactions with the cytoplasmic inclusions: (1) hyperchromatic stage of the Nissl substance; (2) "shaggy," ragged state of the Nissl substance; (3) formation of beaded networks of the Nissl substance, with vacuolation of the structural protein; (4) degeneration of cell bodies, with expulsion of intracytoplasmic particles; (5) shadow cells, and (6) fragmentation of neurons and liberation of particles, with general and perivascular distribution. Glia reactions in fiber bundles, amyloid bodies and patches of degeneration of the myelin sheaths, with large glia cells, were also present. Another striking feature was the increase of the intercellular protetin framework among the glia cells.

Hyperchromatic reactions of the nerve cells were common and represented the first stage of formation of the inclusion bodies. Many neurons which contained these bodies appeared normal at first sight (fig. 1 *A*). In such neurons large blocks of Nissl substance were often crowded into the periphery of the cell, so that the wall bulged. Near the middle the cell might present a clear, and somewhat more finely granular, chromidial mass (*i*), at one side of the nucleus. It was this mass which contained the colony of inclusions. Around this mass larger blocks of Nissl substance showed evidence of abnormal distribution, with the resulting bulging at the surface. Rupture of the cell surface was rarely seen at this stage.

Granular cytoplasm and the shaggy, tattered appearance of the surface of the neuron (fig. 1 *B*) was the result of crowding of the Nissl substance into the cell wall in an irregular way. This ragged outline of Nissl substance at the surface of the cell almost always indicated the presence of a large number of inclusion bodies in the cell, often seen as a more finely granular area (*i*). Sometimes the granules projected from the surface.

At a later stage changes in Nissl substance became conspicuous. As the neuronal disease advanced the nerve cells took on a diffuse stain (fig. 2 *C*), and the Nissl substance was altered to a variable degree until it became arranged in an irregular, beaded network. The interstices of this network appeared as somewhat clear vacuoles (fig. 2 *D*). As the neuron became impoverished in Nissl substance, it became smaller and the vacuolar appearance more prominent.

At this stage the beaded, irregular contour of the cell wall was striking. The dendrites, at their point of departure, showed irregular, beaded enlargements (fig. 2 *D*), so that many neurons appeared distorted, with gnarled and twisted dendrites. In some cells dendrites appeared to be reduced and were often lacking. The structural protein, with its various deformations, was conspicuous.

The nucleus of the cell did not seem to be affected, though it was usually swollen and in time was displaced toward the periphery (fig. 2 *D*). The nucleolus was prominent and usually swollen. The position of the nucleus in hyperchromatic cells might be nearly central; in other stages, when the cell was occupied on one side by a large colony of the inclusion bodies, it was eccentric (fig. 3).

Bars of substance which stained like the organisms could be seen on the nuclear membrane of some of the cells. Usually one of these bars, and sometimes two or three, could be identified. In some instances the bars seemed to be inward folds of the nuclear membrane. Some appeared to be thrust into the nucleus, being directed toward the nucleolus. The bars as a rule appeared on the side of the nucleus against which the colony of inclusions was pressed.

Impoverished neurons might persist as weakly staining shadow cells (fig. 2 *E*). The impoverishment affected notably the cell body. The Nissl substance became reduced to a faint network, and the cell wall was broken down. Skeletons of shadow cells could be seen in great numbers. Such degenerated neurons probably did not function but seemed to survive in a modified form, with atrophic dendrites. Often only a nucleus, with shreds of cytoplasm, was seen in the cell space.

Dissolution of shadow cells was a striking feature of microgyria. Sometimes only the nucleus and fragments of structural protein could be seen (fig. 2 *F*). Shriveled and atrophic dendrites could be observed as small stumps. Neuroglia cells lay close by, and phagocytosis may have been taking place. Cell debris and amorphous products of degeneration were common. These were sometimes globular. Shreds of cell walls might be numerous. Other cytoplasmic and neuroglial changes which were noted have been described so often by other investigators that they need no elaboration here.

Scars appeared in the areas where neuronal degeneration was extensive and complete. Various fragments of cell debris were seen in these scars; neuroglial replacements predominated. This is a picture familiar to the neuropathologist. The isolated clusters of inclusion bodies which occurred in the neurons, as well as in the scars

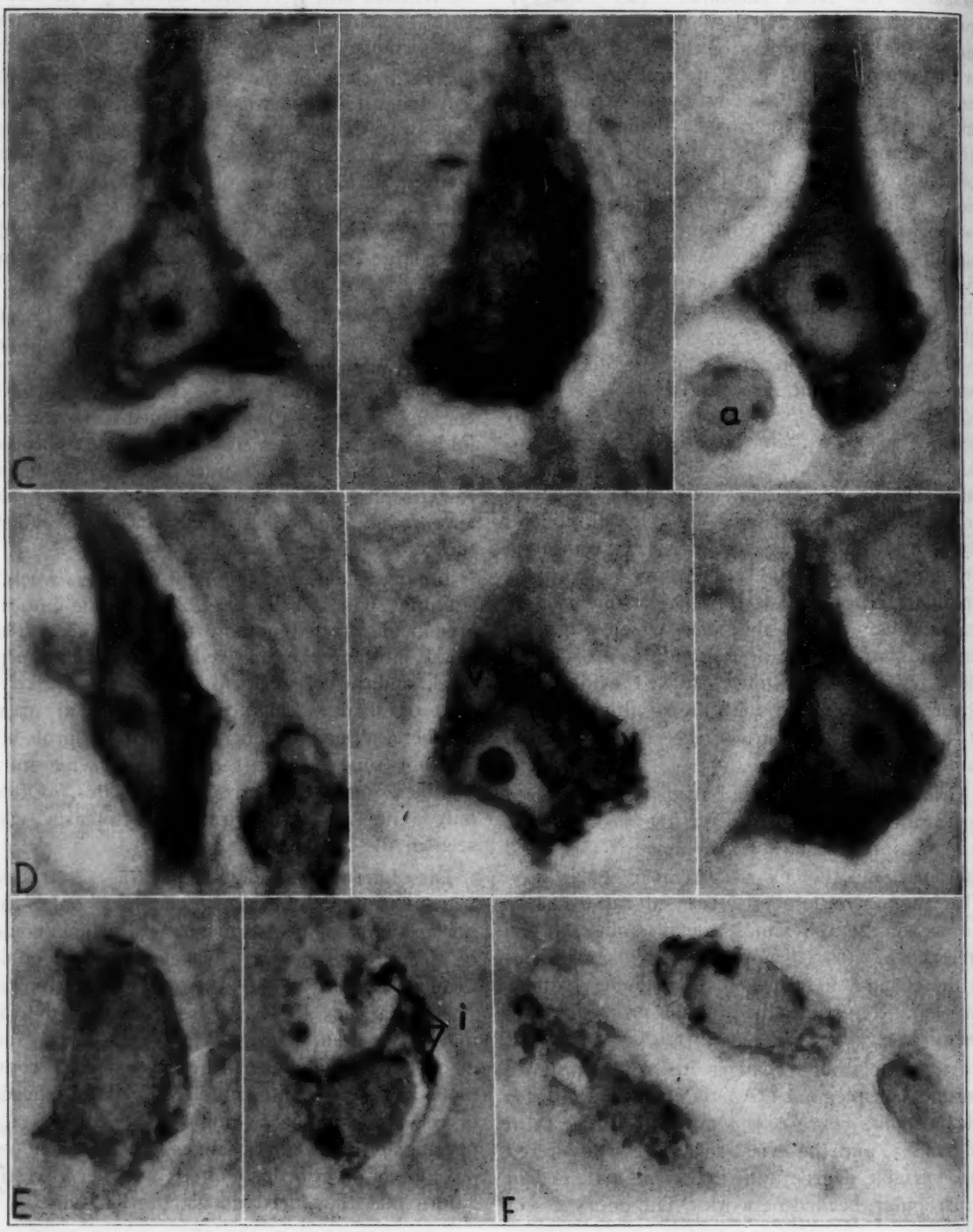


Fig. 2.—Advancing stages in degeneration of neurons in a case of postencephalitic paralysis agitans, as shown by the ordinary cresyl violet stain. *C* shows irregular beaded networks of Nissl substance; *D*, formation of vacuoles (*v*), as well as blisters on the cell surface; *E*, cells with broken-down cytoplasm, and *F*, shadow cells, often with the nuclei alone remaining. The nucleus of an astrocyte is shown at *a* and inclusion masses appear at *i*. Magnification, 2,475.

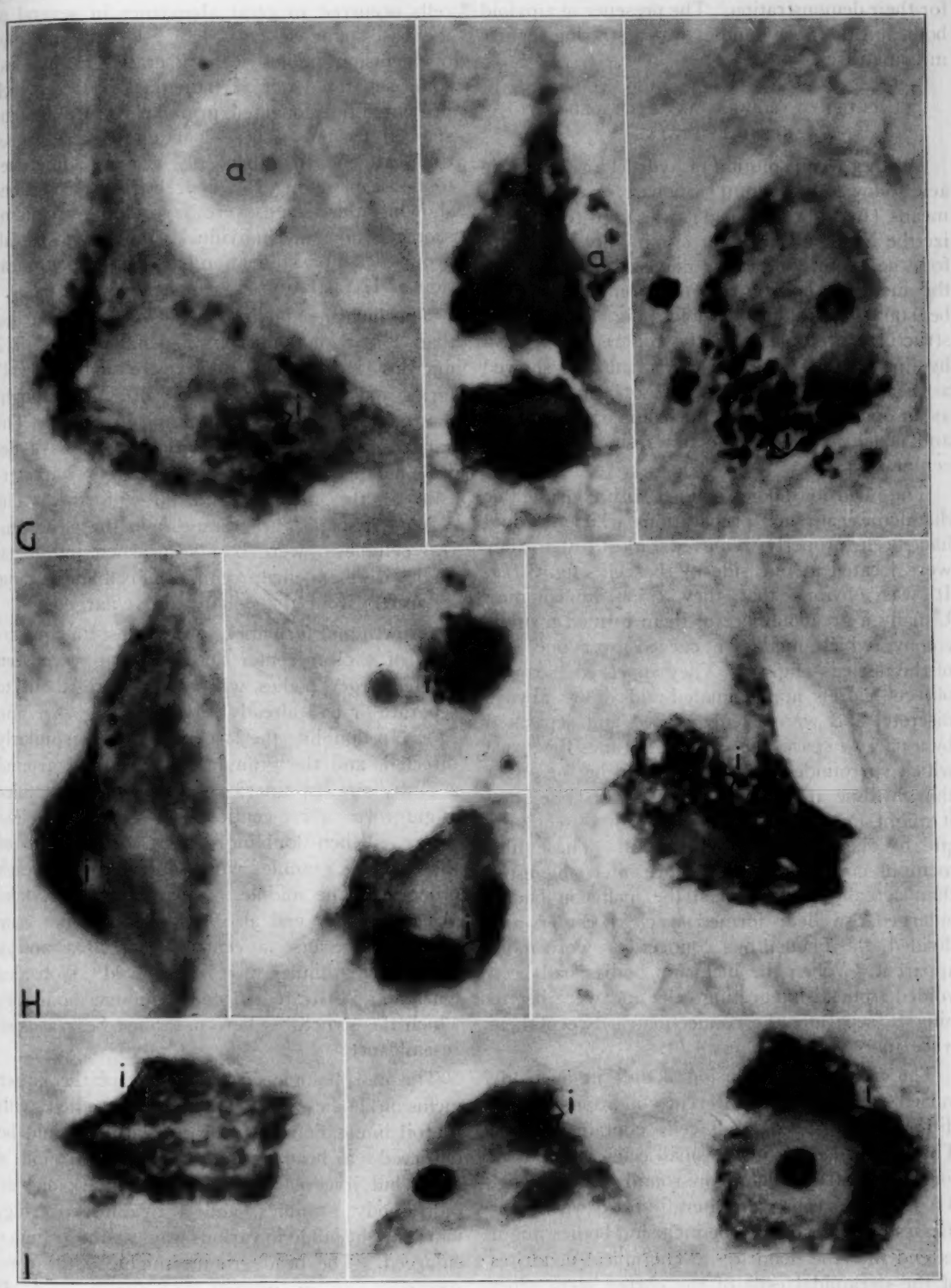


Fig. 3.—Neurons in cases of postencephalitic paralysis agitans, showing inclusion bodies (*i*) as dark objects, as they appear when stained by the special citric acid–sodium carbonate–cresyl violet method. The nucleus of an astrocyte is indicated by *a*. × 2.475.

and around blood vessels required a special stain for their demonstration. The presence of amyloid bodies in the fiber bundles was a striking feature in some cases.

INCLUSION BODIES AS REVEALED BY SPECIAL STAINS

When a weak solution of hydrochloric or citric acid followed by sodium carbonate, or some other means (hydrobromic acid), was used to neutralize the Nissl substance, or else modify its affinity for the stain, and to remove the lipid from the inclusion bodies, the inclusion bodies could be stained with cresyl violet (fig. 3 *G*). The structural protoplasm of the cell remained as a hyaline shadow; the nuclear membrane showed as a faint line; the nuclear protoplasm appeared as an opal mass, and the nucleolus was indistinct and usually swollen, sometimes showing a clear center.

The inclusion bodies stained with considerable distinctness in such preparations when the lipid had been properly removed; most often they were located at one side of the cell (fig. 3 *H*). It was obvious that they grew in colonies. Usually a great number of them formed a single colony, which might be curved over one side of the nucleus (fig. 6). They appeared as round objects, closely approximated and almost always discrete, and were evenly placed and separated by a narrow space (fig. 3 *I*). At times the space which surrounded each body could be seen as a thin hyaline membrane around each one, and it might form filamentous connections from the end of one inclusion to the next. These filamentous connections were more often suggested than actually seen. When the inclusion bodies enlarged, swelled, formed a clear center and divided, the filamentous connections were more apparent. When the inclusion bodies had been voided from disintegrating cells and were beginning to separate, the connections between them were more evident (fig. 4 *J*).

The number of inclusion bodies in a colony varied greatly under different conditions and in different areas. Not all cells contained them. They might be few in the small cells, where they formed small crescents or round masses (fig. 4 *K*). Other cells were literally filled with great masses of inclusions. The inclusion bodies might extend into the dendrites. The apical dendrites of the pyramidal cells of the cortex were often so loaded that they appeared distended and variously deformed so that the cells had a gnarled appearance (fig. 4 *L*). The colony in the cell was not favorable for study of the morphologic character of the inclusion bodies.

Expulsion of the inclusions from disintegrating cells occurred in great abundance in several of the cases studied (fig. 4 *J*). Great quantities of inclusion bodies were liberated in masses; some separated out as individual bodies, which presented the elongated, granular form, and some showed fission. These wanderers were sometimes attached to other cells or were sometimes to be seen among the fiber bundles. Sections showing this stage were favorable for microscopic study of individual bodies and small groups. An oil immersion lens with a magnification of 120 is required.

The inclusion bodies ranged in size from less than 1 to 3 or 4 microns in length and 1 or 2 microns in diameter. Some were small "larval" forms and took a nearly uniform stain throughout, while others were large and granular. The various sizes seemed to represent stages of development. The granular content was characteristic of the medium and large forms. The elongated shape, the granules and the clear central cytoplasm were best seen when individual bodies were expelled from a dead cell and became separated from the colony and enlarged.

Growth and formation of granules, the appearance of a clear center and doubling were seen in the larger bodies when they were about to separate or had already separated (fig. 4 *K* and *M*). In doubling the larger ends were similarly directed, and the granules were spread around a clear, swollen interior. The single granules might form a crescentic curve around a clear center. When doubling occurred, the curves of the dividing granules were separated from each other near the middle. Separation might take place from one end also. Doubling is not easy to follow because a central clear area and a number of granules may be involved. Gonzalez Guzman [2] illustrated in ependymomas bodies to which the structures here described bear a close resemblance.

The morphologic characteristics of the larger inclusion bodies were extensively studied with the oil immersion lens. Four features could be observed: a head granule disposed around a clear, but irregular, cytoplasmic center; one or more body granules, and a hyaline covering, which might bulge in various ways as the granules enlarged. The head granule might be the conspicuous one in intermediate forms, and all the granules might be diffuse or absent in the

2. Gonzalez Guzman, I.: Estudios citonucleolares de los tumores encefalicos: I. Centrosomas y centriolos de los ependimomas; II. Nucleolos de ependimomas, Bol. d. lab. de estud. med. y biol. **1**:136-152, 1942.

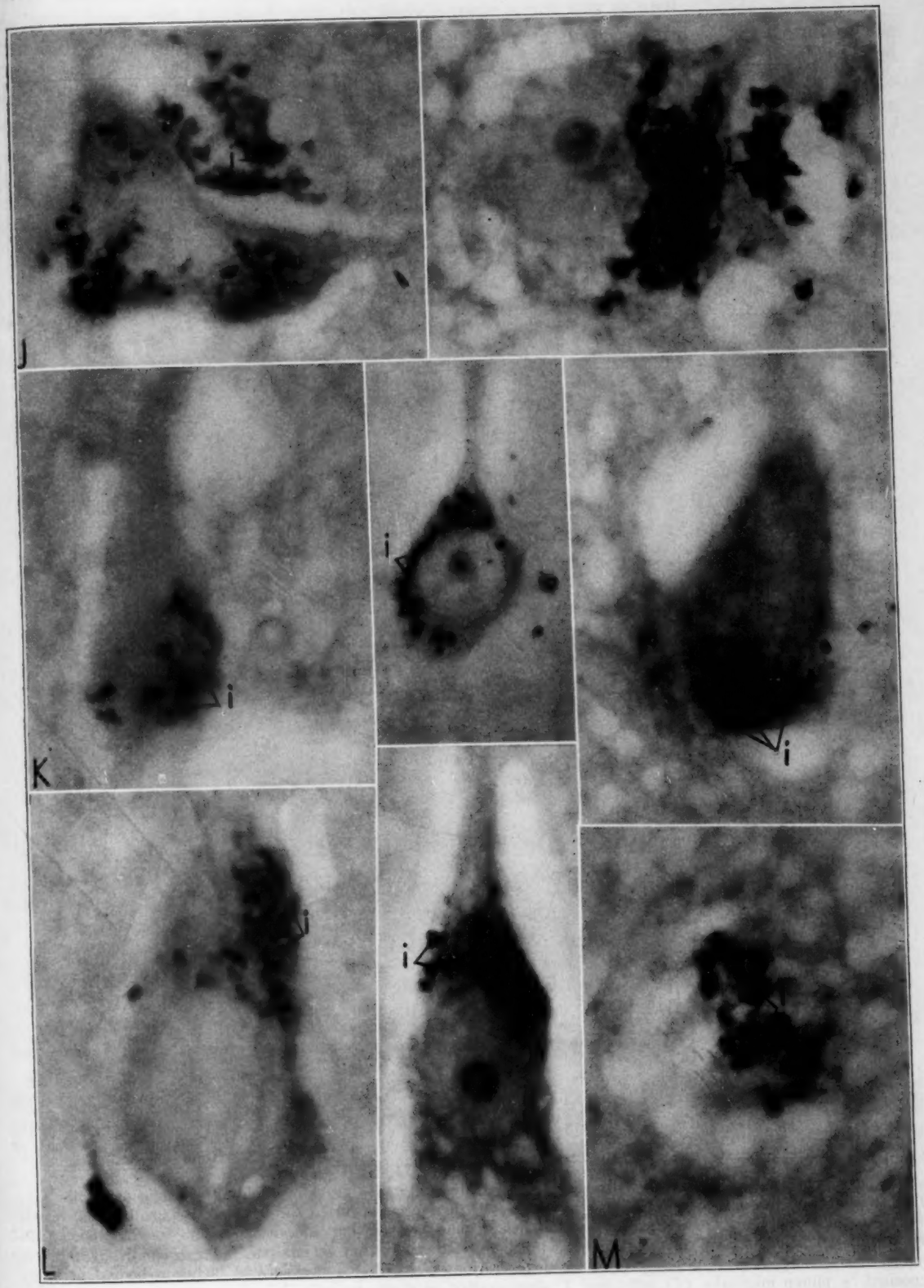


Fig. 4.—Neurons in a case of postencephalitic paralysis agitans, showing various dispositions of inclusion bodies (*i*) in the cytoplasm and other features described in the text. Special stain; about × 2,640.

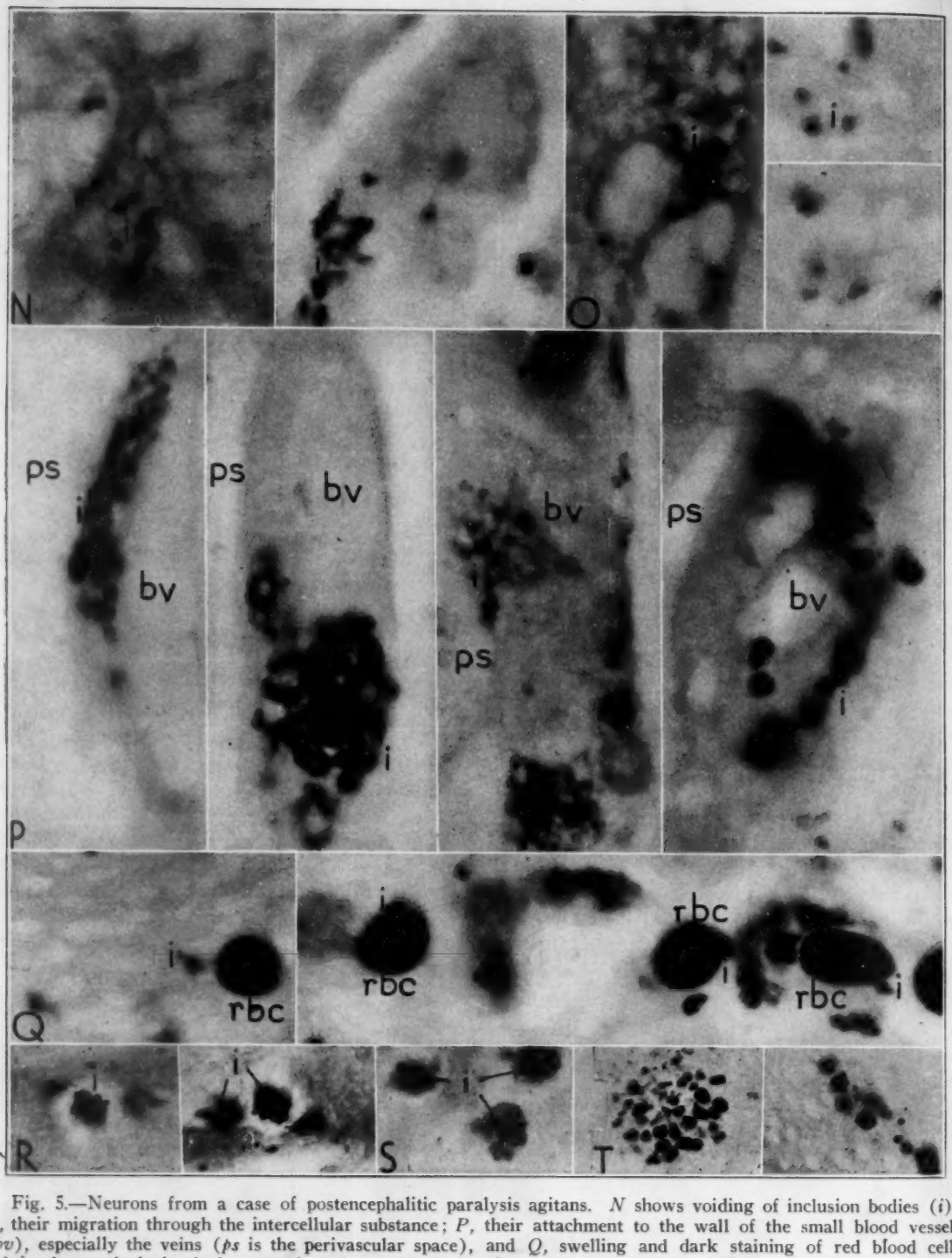


Fig. 5.—Neurons from a case of postencephalitic paralysis agitans. *N* shows voiding of inclusion bodies (*i*); *O*, their migration through the intercellular substance; *P*, their attachment to the wall of the small blood vessels (*bv*), especially the veins (*ps* is the perivascular space), and *Q*, swelling and dark staining of red blood cells (*rbc*) when an inclusion body comes in contact with them. Otherwise they remain pale blue. × 2,475. *R* indicates clumps of inclusion bodies in the process of degeneration; *S*, caches of degenerated inclusion bodies, probably deposited by plump microglia (?) cells, and *T*, degeneration products, including pigment and amorphous material. × 690.

"larval" forms. In larger forms the head granule was often large and, together with the body granules, gave the inclusion body a segmented appearance. This appearance of segmentation was enhanced during the stage of doubling by the clear central cytoplasm.

Large forms sometimes attained a considerable size and appeared segmented, with two or three or four granules. At this stage separation or fission was often present, with a series of three or four granules on each side, partially or wholly separated in a longitudinal direction. The granules were spread around a clear center, giving the appearance of a ring formation during the process of separation, much as was illustrated by Gonzalez Guzman.[2]

When the inclusion bodies were voided from the nerve cells, they became entangled in the intercellular substance. This was formed by a fenestrated protoplasmic fabric which filled the spaces around and between the glial framework. The Holzer stain revealed that the glia cells formed only a small part of the intercellular substance. As the nerve cells were lost, the intercellular substance seemed to fill up the space which was vacated.

At times the cytoplasm of disintegrating cells seemed to be in continuity with the fenestrated intercellular substance (fig. 5 *N*). Many groups of voided inclusion bodies could be seen in the intercellular substance, through the spaces of which they seemed to migrate in mass or singly by some process. They were to be seen both near and far from their cells of origin (fig. 5 *O*). Some ultimately reached the small neighboring blood vessels. The microglia cells did not seem to be the chief carriers of the inclusion bodies.

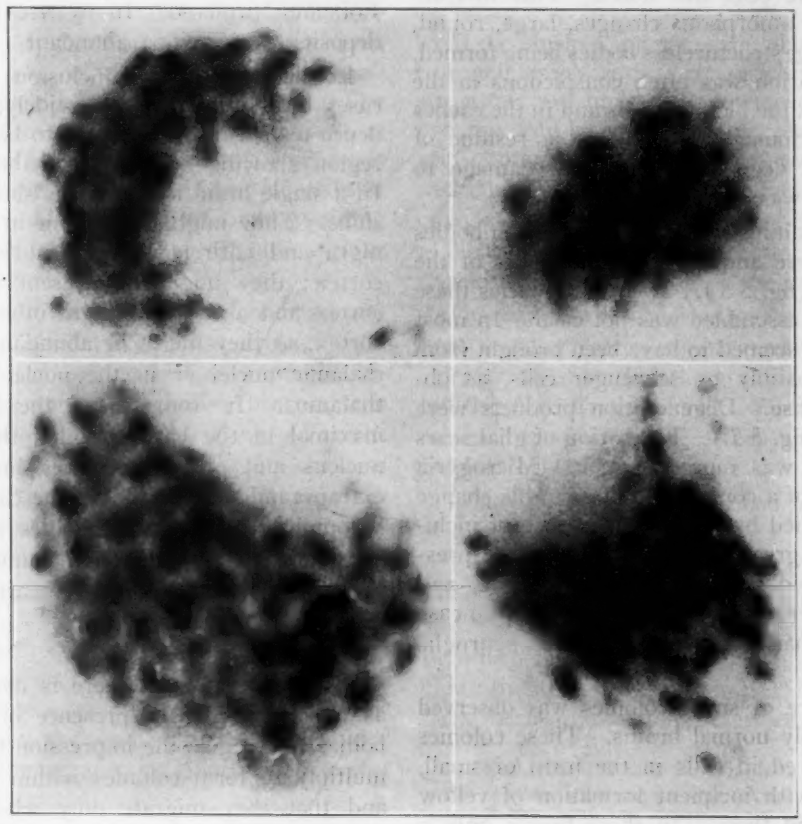


Fig. 6.—Four colonies of inclusion bodies from cells of the lateral hypothalamic nucleus in a case of pituitary basophilism. The cells were in the tattered stage. Special stain; × 5,120.

The association of inclusion bodies with the small blood vessels, especially the veins and capillaries, was a striking and common picture following cellular breakdown and voiding of inclusions (fig. 5 *P*). The inclusion bodies were embedded in the outer walls of the blood vessels, becoming intracytoplasmic. A few could be seen in the microglia cells. They were not observed in the outer meninges. When stained for fat, they appeared as granular fatty masses. The special stain employed in this study showed the

inclusions loosely arranged in the wall of the blood vessel. Here they swelled and took a darker stain, and the hyaline membrane could be clearly seen. The outer wall of the vessel was thickened and the lumen was often constricted. Local escape of red blood cells was rarely seen.

Long search showed that the inclusion bodies rarely entered the lumen of the blood vessel, and then usually singly. When a single inclusion came in contact with a red blood cell, the latter became globular and took on an intense stain with cresyl violet (fig. 5 *Q*).

Degeneration of the inclusion bodies expelled from the nerve cells was noted. This was accompanied by amorphous changes, large, round, darkly staining, structureless bodies being formed. This degeneration was often conspicuous in the masses around the blood vessels and in the caches (fig. 5 *R*). Some bodies showed a residue of pigment after degeneration. Local damage to blood vessels was considerable.

Caches containing degenerated inclusion bodies were noted here and there among fibers of the white matter (fig. 5 *S*). By what agencies these deposits were assembled was not clear. In most instances they seemed to have been brought from a distance, possibly by scavenger cells, as observed in 1 case. Degeneration products were also present (fig. 5 *T*). Formation of glial scars in the cortex was more common. Microgyria was evidence of a congenital onset. This change was accompanied by a heavy incidence of inclusion bodies, a great loss of nerve cells, the presence of amyloid bodies in the fiber tracts and glial replacements in atrophic cortex. In no case was this primarily a disease of the neuroglia cells.

The presence of small colonies was observed in 2 presumably normal brains. These colonies were sequestered in cells in the form of small, round cysts, with incipient formation of yellow pigment around them. In these brains only a few cells were affected; the majority did not show inclusion bodies. The pigment appeared to be derived from the degeneration of inclusion bodies and was apparently not a product of degeneration of the nerve cells, which was also present in some instances. Usually the pigment was related to the inclusions in intracellular, as well as perivascular, situations. It is not possible to say that all the dark pigment in cells of the substantia nigra and the locus caeruleus was of this origin.

The formation of yellow pigment around the colonies of inclusion bodies was not seen in cases of chronic disease. Dark pigment was conspicuous in the cases of paralysis agitans. All cells were not affected—only those which contained the inclusions. In such cells the inclusions appeared to be sequestered by a membrane within the cell body. Such masses of inclusions might show encrustation of pigment over their ends, giving a punctate appearance. Yellow pigment was seen in some of the hypothalamic cells. It was less frequent in cells of the thalamus and was rare in cells of the cerebral cortex. Dark pigment was most notable in the substantia nigra. The pigmentary changes associated with degeneration of neurons in the substantia nigra, the basal ganglia and other regions in paralysis agitans were well illustrated and described by Foix and Nicolesco.[3] In my cases the pigmentary deposits were not so abundant.

Localization of the inclusion bodies in the cases here studied varied widely, but the incidence tended to be local and to be limited to the region showing other neuropathologic changes. In a single brain not all areas showed the inclusions. They might be present in the substantia nigra and entirely (?) absent in the cerebral cortex; they might be present in the frontal cortex and almost absent in other parts of the cortex, or they might be abundant in the hypothalamic nuclei or in the nuclei of the dorsal thalamus. In some cases the incidence was maximal in the basal ganglia, the subthalamic nucleus and other structures linked with the extrapyramidal system. In the case of pituitary basophilism the inclusion bodies were most numerous in the lateral hypothalamic nucleus, and they were also noted in the heart muscle.

COMMENT

With proper stains there is never any doubt as to the identity or presence of the inclusion bodies. One gets the impression that the bodies multiply to form colonies within the cytoplasm and that they migrate only when damage or degeneration of the cell takes place. This indigenous nature of the inclusion bodies, in what would pass as fairly healthy neurons, suggests a symbiosis, for when voiding or migration is absent the bodies are strictly intracytoplasmic and are rarely detected outside the cell.

It can also be shown that the intracytoplasmic bodies are not general components of all nerve cells. Only a small proportion of the cell population may be involved, and in 3 brains no cytoplasmic inclusions were observed. In 1 brain

3. Foix, C., and Nicolesco, J.: Anatomie cérébrale les noyaux gris centraux et la région mesencephalo-sous-optique, Paris, Masson & Cie, 1925, p. 581.

sequestered inclusions were notetd in some cells of the subthalamic and tegmental regions, but none in the cortex.

In healthy-looking cells with inclusion bodies, the Nissl substance stains strongly, and large hyperchromatic blocks occur near the surface, so that the appearance of abnormal distribution is produced. The "shaggy," ragged stage is also indicative of the presence of inclusion bodies. Diffuse staining of the Nissl substance marks the beginning of cell degeneration. The formation of vacuoles and the blister-like distortion of the interior and the surface of the cell, together with the reticular distribution of faintly staining Nissl substance, indicates an advanced stage of neuronal degradation. It is hard to believe that such neurons could function normally. This is particularly true of the shadow cells. Pericellular satellitosis indicates neuronophagia. The microglia cells seem to be active when there is considerable cell debris. The over-all picture is one of initial symbiosis and, later, of cellular degeneration and fragmentation, with voiding of inclusion bodies. The inclusion bodies survive and outlive a dead cell.

The nature of the inclusion bodies is an unsolved problem. That they pass through successive stages of growth, development and multiplication by fission, or a similar process, can be affirmed from many observations. Various staining reactions indicate that their chemical composition is some protein organization which differs from that of other cell components, even the nucleolus. Their reaction is strongly basophilic for their own granules. They have a rich lipid content. Their morphologic character remains constant for the various stages of their growth and fission. They also degenerate, becoming amorphous and pigmented. Fission is seen best when the bodies have been voided from cells and have taken an elongated, granular form.

The intracytoplasmic inclusions are not artefacts, nor are they products of neuronal degeneration itself. When cell destruction has occurred, there is of course much cell debris. One must be familiar with the objective facts to appreciate fully the validity of these statements.

The Golgi apparatus resembles these bodies in an uncertain way. It is true that the Golgi apparatus lies against the side of the nucleus, that it forms in clusters, that it disintegrates and that it takes on a varied morphologic appearance. But the nature of the Golgi apparatus is still undetermined, and its morphologic character is protean (Kirkman and Severinghaus [4]). In the absence of direct comparison, it is not possible to draw an analogy between the inclusion bodies and the Golgi apparatus at this time. However, it is not likely that the inclusion bodies represent a hypertrophy or a pathologic development of the Golgi apparatus, or of any other substance which may be considered as a normal growing constituent of the nerve cell. In the literature there is no statement that the Golgi apparatus is associated with neuronal degeneration.

The resemblance of the inclusion bodies to microsomes, described by Claude,[5] is suggested on study with low magnifications. This author found the microsomes in a variety of tissues, including the liver, testis, kidney and muscle, from the chicken, mouse, rat, rabbit, guinea pig, cow and amphibians. He concluded that these structures are built on a framework of nucleoproteins, and he suggested that, like other nucleic acid–containing structures, they may be endowed with the power of self reproduction. His discussion of the chemical nature of the microsomes is illuminating and may be pertinent here if they should prove to be related to the inclusion bodies.

The bodies described by the Scharrers as neurosecretory droplets may come in for consideration.[6] Some of the granules demonstrated by these authors look like microsomes, while others resemble the secretory granules distinguished by Claude.

Long ago Herrick [7] described inclusion bodies in a case of dementia paralytica.

Brumpt[8] stated the case for Encephalitozoon rabiei, which Manouélian and Viala [9] described, and there seems to be a resemblance of this organism to the inclusion bodies described here. Since the work of Goodpasture,[10] inclusion bodies have been most often described in connection

4. Kirkman, H., and Severinghaus, A. E.: A Review of the Golgi Apparatus, Anat. Rec. **70**:413-431 and 557-573, 1938; **71**:79-103, 1938.

5. Claude, A.: Distribution of Nucleic Acids in the Cell and the Morphological Constitution of Cytoplasm, Biol. Symposia **10**:111-129, 1943.

6. Scharrer, E., and Scharrer, B.: Secretory Cells Within the Hypothalamus, A. Research Nerv. & Ment. Dis., Proc. (1939) **20**:170-194, 1940.

7. Herrick, C. L.: Report upon Pathology of a Case of General Paralysis, J. Comp. Neurol. **3**:141-162, 1893.

8. Brumpt, E.: Precís de parasitologie, Paris, Masson & Cie, 1927, p. 378.

9. Manouélian, Y., and Viala, J.: Parasite of Hydrophobia, Ann. Inst. Pasteur **38**:258-267, 1924.

10. Goodpasture, E. W.: Cellular Inclusions and the Etiology of Virus Diseases, Arch. Path. **7**:114-132 (Jan.) 1929.

with virus diseases. Rivers [11] gave a good summary of this subject, with an excellent bibliography. The encephalitozoon infection in laboratory animals recently described by Perrin [12] seems, also, to be similar. The illustrations by Weiss [13] show rickettsial bodies, the size and configurations of which are similar to the inclusion bodies here described. Marburg [14] described inclusion bodies in spinal ganglion cells in cases of infantile amaurotic family idiocy and gave an excellent discussion of the literature on inclusion bodies associated with nervous diseases.

SUMMARY

In primary neuronal degeneration associated with formation of inclusion bodies, six stages are recognized by the ordinary staining methods: (1) hyperchromatic reaction, with blocks of Nissl substance in the periphery of the cells; (2) "shaggy," ragged state of the Nissl substance, with irregular cell outline; (3) formation of vacuoles and blisters, with deformation of cells and poverty of Nissl substance; (4) degeneration of cells, voiding of inclusion bodies and pericellular satellitosis; (5) shadow cells and cell fragments, a stage which the inclusion bodies survive, and, (6) degeneration of sequestered colonies in the cell, with punctate formation of pigment. The absence of inflammatory reactions is noteworthy.

The inclusion bodies may be revealed by treating material fixed in a solution of formaldehyde with solutions of citric acid, sodium carbonate and cresyl violet. The chemical composition of these bodies differs from that of other constituents of the nerve cell. The inclusion bodies are discrete, punctate, round or elongated objects, which grow in colonies within the cytoplasm. They are 1 micron or less in diameter and 2 to 3 microns in length. Several stages of their growth have been observed. Some are small, agranular "larval" forms; some are of medium size, and some are large. The larger forms show an internal granular, stainable structure, with a clear central area and one or more granules behind a head granule. A membrane surrounds the individual bodies and holds them together. Fission, resulting in doubling, has been seen in the larger forms. As the neuron degenerates the inclusion bodies are voided from the cell in masses, which pass to the blood vessels or form deposits elsewhere. Many individual bodies separate from these masses and are strewn about in the nerve tissue. Atrophy of the cortex, neuroglial scars, deposits of gitter cells, sometimes around blood vessels in areas close to softenings, amyloid bodies and other pathologic lesions mark the terminal process. Punctate formation of yellow pigment is seen when the bodies degenerate within the nerve cells. When the cluster voided from a cell degenerates, pigment is sometimes seen in its place.

The growth and behavior of the inclusion bodies indicate that they are living things, not artefacts, degeneration products or a normal constituent of the nerve cell. The name inclusion bodies does not seem appropriate. If they prove to be parasites, the term encephalitozoon might be better. That they react strongly to reagents containing citric acid, sodium carbonate or bromine is worthy of note. That they accumulate around blood vessels, with production of local damage, is a striking observation.

METHOD OF STAINING OF INCLUSION BODIES IN NEURONS

The special technic described here was devised by Miss Elly Henschel, to whose industry its perfection and application are due. The procedure is suitable for material fixed in a solution of formaldehyde.

1. Blocks of tissue a few millimeters in thickness are rinsed in tap water for one to two hours and then in distilled water for about one hour.

2. The blocks are then transferred into a 1 per cent aqueous solution of citric acid and placed on the electric plate, at a temperature of 48 to 50 C., for three hours.[15]

11. Rivers, T. M.: Viruses and Virus Diseases, Stanford University, Calif., Stanford University Press, 1939, p. 133.

12. Perrin, T. L.: Spontaneous and Experimental Encephalitozoon Infection in Laboratory Animals, Arch. Path. **36**:559-578 (Dec.) 1943.

13. Weiss, L. J.: Electron-Micrographs of Rickettsiae of Typhus, J. Immunol. **47**:353-357, 1943.

14. Marburg, O.: Inclusion Bodies and Late Fate of Ganglion Cells in Infantile Amaurotic Family Idiocy, Arch. Neurol. & Psychiat. **49**:708-723 (May) 1943.

15. In step 2 the solution of citric acid may be replaced by a 1 per cent solution of citric acid in 70 per cent alcohol, a 1.5 per cent solution of hydrochloric acid in 70 per cent alcohol, a 6 to 8 per cent aqueous solution of hydrobromic acid (chloride free) or a 1 per cent aqueous solution of tannic acid. Ordinary paraffin sections may be brought from distilled water into a 1 per cent aqueous solution of sodium citrate and placed on the electric plate, at a temperature of 50 C., for three to four hours. They are then washed quickly in distilled water and treated as indicated in items 8 and 9.

The method can be used also for frozen sections, as well as for material embedded in pyroxylin (after the pyroxylin has been removed). Instead of cresyl violet, the Giemsa, Jenner or May-Grünwald stain may be used. Treatment with sudan III brings out the lipid content of the inclusion bodies.

3. They are washed three times in distilled water for five minutes, brought into a 1 per cent aqueous solution of sodium carbonate (C.P.) and again placed on the electric plate, at a temperature of 40 C., overnight.

4. The blocks are washed in distilled water two to three times.

5. They are then transferred into 80 per cent alcohol for a few hours and then into 95 per cent alcohol, absolute alcohol and chloroform and are embedded in paraffin.

6. Paraffin sections are cut 3 to 5 microns in thickness.

7. The sections are brought through toluene (reagent), absolute alcohol, 95 per cent alcohol and 70 per cent alcohol into distilled water.

8. Sections are placed in a 0.25 per cent aqueous solution of cresyl echt violet in the oven for about twenty minutes.[16]

9. Sections are washed for a few minutes in distilled water and destained in 70 per cent alcohol. In 95 per cent alcohol the stains turn blue. Dehydration is completed in absolute alcohol, and the sections are cleared in toluene (reagent) and mounted in clarite.

The Sarah Manning Sage Endowment Fund aided in the technical study.

Department of Zoology, Cornell University.

16. Prepared by Coleman & Bell Company, Norwood, Ohio.

Clinical Notes

ROENTGENOGRAPHIC EVIDENCE OF RELIEF OF CHRONIC INCREASED INTRACRANIAL PRESSURE FOLLOWING A TORKILDSEN OPERATION

ERIC OLDBERG, M.D., CHICAGO

The case to be reported demonstrates the reversal of a process familiarly observed in cases of increased intracranial tension of long standing. As is well known, when tension remains above normal the greater part of the time for six months or more, the inner table of the skull becomes indented as a result of the pressure atrophy caused by the impingement of the cerebral convolutions on it so that markings are visible in the roentgenogram. Thus, in adult life the skull may take on the appearance of "digital" or convolutional markings, which is normal in childhood and adolescence.

In 1939 Torkildsen [1] devised a procedure for relief of chronic increased intracranial tension due to atresia or neoplastic occlusion of the aqueduct of Sylvius. Essentially, the operation consists in inserting one end of a small rubber catheter through an occipital burr hole into the lateral ventricle and carrying the other end of the catheter, by way of a small suboccipital decompression opening, into the cisterna magna. The fluid formed in the lateral and third ventricles is thereby enabled to by-pass the aqueduct of Sylvius and get into the subarachnoid space.

In the case to be described, roentgenograms of the skull taken prior to this procedure showed pronounced convolutional atrophy. The Torkildsen procedure was performed, with complete symptomatic relief of pressure and with recalcification of the inner table of the skull and consequent disappearance of the pressure markings. This recalcification and obliteration of the pressure markings were far too obvious and spectacular to be accounted for by the comparatively slight increase in the age of the patient and must be assumed to be due to a reversal of the process of pressure atrophy, such as occurs under similar circumstances in other portions of the skeleton. The case therefore demonstrates visibly, as well as symptomatically, the possible efficacy of the Torkildsen procedure and is deemed worth reporting in view of the relatively rare opportunity of making such a roentgenographic observation.

From the Department of Neurology and Neurological Surgery, University of Illinois College of Medicine, and the Illinois Neuropsychiatric Institute.

1. Torkildsen, A.: Acta psychiat. et neurol. **14**:221, 1939.

REPORT OF A CASE

History.—D. W., an unmarried girl aged 19, entered the Illinois Neuropsychiatric Institute on May 12, 1942, having been referred by Dr. Elmer S. Horine, of Maywood, Ill. According to the history, the patient had been "nervous and excitable" for three years, and during her excitable episodes various vasomotor phenomena appeared, manifesting themselves chiefly as white patches on the skin. One and a half years before admission there developed a fine tremor of both hands, most marked in intentional movements. The patient first observed it when eating and afterward noticed it whenever she performed any voluntary act with the upper extremities. She stated the belief that when she was at rest this tremor was absent. One year before admission the patient noticed also a tremor of the head, which she felt was steadily increasing. Again, this tremor disappeared when the patient was relaxed.

Examination.—The patient had syndactylism of the proximal phalanges of the second and third toes of both feet. There was also a bilateral "spade thumb," with some webbing of the fingers of both hands, the right hand being involved to a lesser degree than the left. The blood pressure was 134 systolic and 70 diastolic. There was a fine tremor of the head, estimated at 200 to 300 excursions per minute, which was constant when the patient was erect and was exaggerated by excitement. There was also a tremor of both hands, more pronounced on the left side and exaggerated in intentional movements. During voluntary movements the tremor seemed to extend to involve the trunk and the right lower extremity. There was mild bilateral optic nerve atrophy, with a hazy margin of the right disk. Lumbar puncture yielded clear, colorless fluid, under an initial pressure of 190 mm. of water. Roentgenograms of the skull revealed pronounced thinning, separation of the sutures, convolutional atrophy and the typical thinning of the sella seen with generalized chronic increase in intracranial pressure.

Although an initial clinical impression of possible hepatolenticular degeneration was created, it was felt, in view of the slightly elevated spinal fluid pressure and the appearance of the roentgenograms of the skull, that a ventriculographic study should be made. This accordingly was done on May 19, an enormous internal hydrocephalus, involving the lateral and third ventricles, being disclosed when air was injected, after removal of 360 cc. of fluid. Phenolsulfonphthalein passed freely from one side to the other.

Operation.—On May 21 the posterior fossa was explored by Dr. W. A. Gustafson. No tumor was dis-

closed, although there was a pronounced foraminal herniation of the cerebellar tonsils. A Torkildsen procedure was thereon carried out, the right lateral ventricle being connected with the cisterna magna. The immediate postoperative course was stormy, but after about a week the patient's condition began to improve. The tremor also lessened, and she was discharged on June 30.

Subsequent Course.—After her discharge from the hospital, the patient continued to improve and was employed for a year and a half as an "order picker" by a large mercantile company, it being her duty to fill orders as they came in. The plant physician stated that the patient's foreman considered her one of the best workers in his department and said she was well liked and had a fine personality. According to the patient's family, she was fairly bright during this period, wrote good letters to her brother and often "wisecracked." She continued to work and remained well until Feb. 22, 1944, when she had a cold and complained of pain in the back of her head and frontal headache. On February 26 she had diplopia, and about 2 o'clock p. m. she became somnolent. She vomited once, refused food and only occasionally answered, with a monosyllabic "yes" when she was spoken to.

She was readmitted to the Illinois Neuropsychiatric Institute on February 28. She responded only to noxious stimuli. The blood pressure was 116 systolic and 80 diastolic. There was a fine tremor of the upper extremities but not of the head. The deep reflexes were everywhere hyperactive, with a sustained ankle clonus on both sides. The Babinski sign was obtained bilaterally. Roentgenograms of the skull showed almost complete disappearance of all digital markings, increase in the thickness of the skull, increased contrast of the frontal sinuses and diminution in size of the formerly expanded and thinned sella turcica (figs. 1 and 2). Lumbar punctures on admission and on the following day

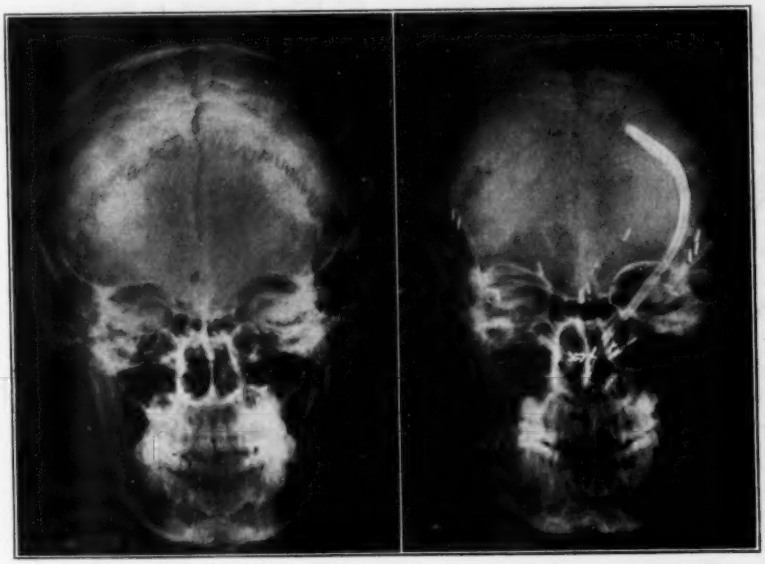


Fig. 1.—At the left is a posteroanterior view, taken May 13, 1942; at the right, a similar exposure, taken by the same technician and with the same apparatus and technic, on Feb. 28, 1944. Note the disappearance of convolutional markings and the thickening of the skull, with resultant increased contrast of the frontal sinuses.

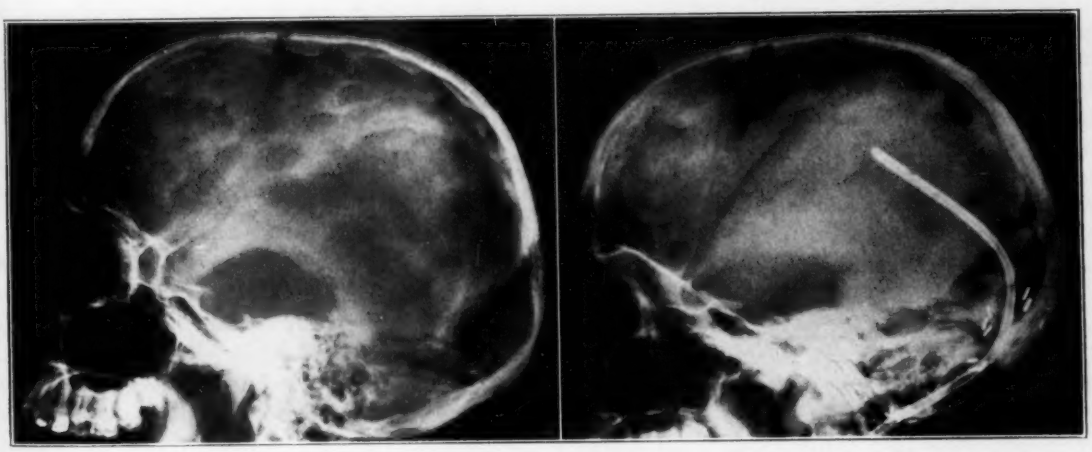


Fig. 2.—Lateral views corresponding to the posteroanterior views shown in figure 1.

yielded pressures of 175 and 240 mm. of water respectively. The patient's condition was temporarily much improved on several occasions by ventricular tapping, the pressure varying from 250 to 350 mm. of water. In an effort to provide a new short circuit of fluid, an opening was made in the third ventricle by Dr. H. P. Maxwell, on March 4. This did not greatly influence the course, however; the patient's condition grew worse, and she finally died, on March 22.

Permission for autopsy was granted, and the essential portion of the gross description by Dr. Percival Bailey follows.

"In the right occipital pole, about 2 cm. to the right of the midline, was a puncture wound, with a smooth, glistening wall, which extended into the tip of the occipital horn of the lateral ventricle. The cerebellum appeared to be normal, and there was no indication of an old operative trauma in that region. Medial longitudinal section of the brain through the corpus callosum and the brain stem disclosed extensive internal hydrocephalus, involving the lateral and third ventricles. The fourth ventricle was of normal size; the aqueduct of Sylvius was occluded."

The catheter when removed was found to be patent, and water passed through its lumen easily. Dr. Bailey's microscopic description of the brain follows.

"Serial sections demonstrated proliferation of the neuroglia, which had almost completely occluded the aqueduct in its middle portion. A small, tortuous ependymal channel was occluded by a blood clot, with old blood pigment in the neighborhood. The gliosis involved the entire thickness of the section at this level. The track of the catheter was clean; there was slight gliosis, and small collections of lymphocytes were present in the immediate neighborhood.

"The impression was that of atresia aqueducti with secondary hydrocephalus."

University of Illinois College of Medicine.

Obituaries

CHARLES WALTS BURR, M.D.
1861-1944

On Feb. 19, 1944 Charles Walts Burr passed away in his apartment at the Drake Hotel in Philadelphia. His death had been expected by reason of the known nature of his illness, but to his host of friends and associates it occasioned great sorrow. His had been a most versatile personality. He was a man of medicine, of science and of letters. He was more than a physician; he was a teacher, an investigator and an author of no mean caliber. He belonged to that generation of nationally known Philadelphia neuropsychiatrists who had added much luster to their specialty for many years, and he was the last survivor of the Dercum-Mills-Lloyd-Spiller group. It has been said of him that he did not know how to play, but no one ever had any doubt that he knew how to think and work. In my many years' acquaintance with him I am certain that he never took a vacation of more than a few days at a time; and, aside from his studies in Europe immediately following his graduation from the medical school of the University of Pennsylvania, he had never traveled extensively. His entire life was devoted to a continual search for knowledge, not only in his chosen field of medicine but in many other branches of human endeavor. Dr. Burr never married, principally because such a course would have militated against the unimpeded pursuit of his insatiable thirst for knowledge. It may indeed be said of him that he was married to his art.

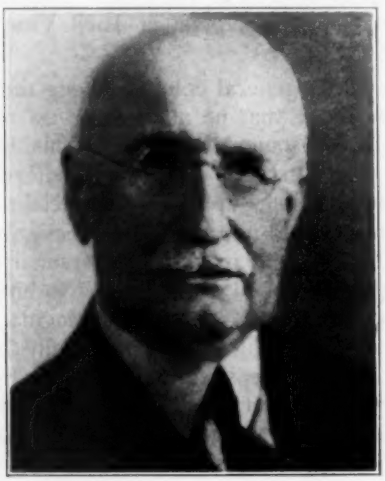
He was a firm believer in the importance of heredity in the predisposition to disease, and this was emphasized in one of his most interesting monographs, entitled "What Is a Good Environment?" He also was a proponent of the organic cause of mental and nervous disorders and was opposed to the more radical phases of the doctrines of psychoanalysis. He prophesied the ascendancy of chemotherapy which has recently come to pass through the miracles of penicillin and the sulfonamide compounds.

Dr. Burr frequently stated to me that his life had been uneventful—no tragedy, but quite a little comedy, in the classic meaning of the word, had come into it. He stated that his ancestors were unimportant, simple, thrifty, and God-fearing, honest people. On his father's side they were English Quakers, who had settled in New Jersey, near Mount Holly, before the Revolution. His mother's people were Irish Presbyterians, named Walts. Aaron Burr was not of Dr. Burr's kin. Charles Walts Burr was the youngest of four children, all sons. His father, Delaplaine Burr, had worked with the one idea of rearing his family comfortably and of giving to each of his children the best in education that Philadelphia offered at the time. Charles Burr was born in Philadelphia, at 1618 Vine street, Nov. 16, 1861.

There was a general opinion among those who knew Dr. Burr that he adhered to no religious belief, and by some he was even considered an atheist. Dr. Burr was a profound believer in an Omnipotent Being, but he held no special religious tenets, and he had no time for the bickerings and theological squabblings that his personal experience and reading had demonstrated to him in relation to the frailty of religious dogma. This attitude toward religion was originally brought into being by an episode in his early life, due primarily to a difference in the religious beliefs of his parents. Up to the age of 10 he had gone regularly to Sunday school at the Arch Street Presbyterian church. One Sunday his regular Sunday-school teacher was ill and his place was taken by an unwise young student of theology, who preached a sermon to the young class on the necessity of baptism in order to obtain salvation. This preacher stated that the unbaptized would be "damned." At this juncture the young Charles arose in his wrath and remarked in a dignified manner, "That is a lie; my father is a Quaker; he has never been baptized, but he will go to heaven as soon as he dies." Whereon he left the church and went home, and from that day Charles Burr never entered another church or evidenced any interest whatever in sectarian religious belief. The incident was an excellent example of how a childhood impression could and did influence the conduct and belief of a person throughout his lifetime.

Dr. Burr was a graduate of the Episcopal Academy, in Philadelphia, of the College Department of the University of Pennsylvania, class of

1883, and of the Department of Medicine of the University of Pennsylvania, class of 1886. He was a college honor graduate and was elected "spoon man" of his class. Shortly after graduation, he was elected to the staff of the Philadelphia General Hospital and became associated with Dr. S. Weir Mitchell as his assistant. He was also associated with Dr. Wharton Sinkler and Dr. William Osler at the Orthopaedic Hospital in Philadelphia, and a little later he studied neurology and psychiatry abroad for two years. Shortly after his return to Philadelphia he became a teacher at Medico-Chirurgical Hospital and College, eventually obtaining the position of professor of neurology at that institution. In 1901 he accepted the chair of psychiatry in the undergraduate school of medicine of the University of Pennsylvania, which chair he occupied



CHARLES WALTS BURR, M.D.
1861-1944

for thirty years, until he was retired "on age," in 1930. He was consultant in neuropsychiatry to many hospitals in and about Philadelphia, and during World War I he was psychiatric consultant to the Surgeon General of the United States Army. In his specialty he was recognized as one of the great leaders in American neuropsychiatry, and in recognition of his eminence in that field he was elected president of the American Neurological Association in 1908. He also had been president of the Philadelphia Neurological Society, of the Philadelphia Psychiatric Society (which he founded) and of the Society of Pathology of Philadelphia, in 1898.

During his life he had collected a large library of scientific books, and in 1929 he gave to the University of Pennsylvania a library of 19,000 volumes. At about that time he was made president of the board of trustees of Episcopal Academy, and in 1933 he was awarded the degree of Doctor of Science by the University of Pennsylvania and was appointed to their board of trustees. He was also a recipient of the Strittmatter medal "for meritorious achievements in the field of medicine" in Philadelphia.

In 1915 Dr. Burr edited and contributed a chapter to "The Textbook on Nervous Diseases" by Hans Curschmann. As an avocation, Dr. Burr was interested in the psychopathology of "genius," and a large section of his library was in reference to men and women of exceptional ability. He disapproved strongly of "mass education," believing that nature did not intend all human beings to have a "higher education"; and his thoughts were expressed in many monographs, one of which was the widely discussed "Broken Minds of Youth."

Dr. Burr was interested in many things other than medicine. He was an active worker in, and at one time president of, the Dickens Fellowship of Philadelphia, the Philosophical Society of Philadelphia and the Franklin Inn Club. The society of Sigma Chi received much of his time and attention. In 1926 he was elected president of the National Eugenics Research Society, and his presidential address was entitled "The Evils of Standardized Education." He also was president of the Numismatic and Antiquarian Society of Philadelphia and of the Medical Club. One of his chief joys was in attending the meetings of the Philobiblon Club. He was instrumental in organizing at the University of Pennsylvania the society known as "The Friends of the Library," the purpose of which was to foster interest in and obtain funds for that library. For many years he had been honorary librarian to the College of Physicians of Philadelphia, and one of the secret ambitions that he had nourished, but never realized, was to become president of that organization. A combination of circumstances defeated that ambition.

During his lifetime, Dr. Burr amassed a considerable fortune in money and in the accumulation of a remarkable library of manuscripts, rare papers and rare books. He was keenly interested in young men who showed promise of special intellectual ability, and in many instances he subsidized the further education of such youths as were financially unable to pursue their quest of medical knowledge. This occurred frequently with students at the University of Pennsylvania and his interns in hospitals and at other institutions in the vicinity of Philadelphia. In this respect, as in the book "Good-Bye Mr. Chips," Dr. Burr might have been likened to Mr. Chips.

in that he had made himself a father to many sons in guiding their youthful footsteps and mental leanings, despite the celibacy of his own life.

Dr. Burr was peculiarly alone during the last twenty years of his life. He had no direct, or even collateral, relatives. The nearest relative that he knew of was a second cousin, who died four years before he did. None of his brothers had married, and he was the last survivor of a really brilliant family line.

Dr. Burr took keen delight in studying and analyzing the mental processes of great authors and personalities in history. His monographs on "Galen," "Jean Paul Marat," "Robert Whytt," "Dr. James and His Medical Dictionary" and "Jerome Cardan as Seen by an Alienist" are a few of his many writings of this nature all of which had intrigued audiences at various medical meetings.

Dr. Burr's philosophy of learning could have been summarized in the words of John Dryden:

"Errours like straws upon the surface flow
But he who would reach for pearls
Must dive below."

He did indeed search "below the surface" for the answer to the riddle of life's voyage. Opinions will vary as to whether his was a complete and well rounded existence, but there is no doubt the world has become a better and more enlightened place in which to live because of the achievements, teachings and guidance of Charles Walts Burr.

FREDERIC H. LEAVITT, M.D.

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Anatomy and Embryology

PIGMENTATION OF OPTIC DISK. DANIEL KRAVITZ, Arch. Ophth. **29**:826 (May) 1943.

Congenital pigmentation of the optic disk is uncommon in the white race, but, according to Oguchi, it is rather frequent in the Japanese. Kravitz describes the fundi in 3 cases and agrees with the opinions expressed by Duke-Elder and Mann as to the source of the pigment, that the pigment is the result of primitive cells of the optic stalk having regressed and developed characteristics of pigment-forming cells in a place where other functions should have developed.

SPAETH, Philadelphia.

PARTIAL AGENESIS OF THE CEREBELLUM AND MEDULLA AND TOTAL AGENESIS OF THE CORPUS CALLOSUM IN A GOAT. W. J. C. VERHAART, J. Comp. Neurol. **77**:49 (Aug.) 1942.

Verhaart gives a clinical and anatomic description of a 1 month old goat with a complex malformation of the central nervous system. The animal was unable to move voluntarily. The pupils reacted to light, and corneal reflexes were present. The goat could not be awakened either by acoustic or by optic stimuli. The malformation consisted of agenesis of the midline areas of the cerebellum, including the entire vermis and the medial halves of the hemispheres. The corpus callosum was absent, and the area of the vestibular and cochlear nuclei was underdeveloped. The clinical symptoms were attributed mainly to this lack of development of the vestibular nuclei.

ADDISON, Philadelphia.

Physiology and Biochemistry

THE EFFECT OF CONTROLLED TEMPERATURES UPON THE SPONTANEOUS ACTIVITY RHYTHMS OF THE ALBINO RAT. LUDVIG G. BROWMAN, J. Exper. Zool. **94**:477 (Dec.) 1943.

Observations were made on 32 female rats of a twentieth generation inbred stock which were confined to voluntary work—registering cages for the first six months of life under conditions of controlled light and temperature.

Daily spontaneous activity rhythm and onset of estrus of normal animals could be modified either by the daily variation of light and dark regardless of environmental temperature, or by the daily variation in temperature when light conditions were constant.

When the temperature was constant and the light varied, the peak of daily activity occurred in the dark interval of each day. With constant temperature and constant darkness, the females tended to persist in the rhythm present when the experiment started. However, when the constant temperature was accompanied by continuous light, females went into a continual series of reversals of their daily activity pattern. When light, or its absence, was a constant factor and the daily temperature varied, the normal females had peaks of activity during the daily cool period.

In bilaterally enucleated animals the daily activity rhythm and the onset of estrus could be controlled by the daily variation in temperature regardless of light conditions. When the temperature remained constant, blind animals were not affected by any variations of light. When the temperature varied, the daily peak of activity of blind animals was correlated with the cool period, and the onset of proestrus occurred at the end of the twelve hour cool period.

The author concludes that the normal nocturnal spontaneous activity rhythm and estrus cycle of the albino rat are dependent on the concomitant daily variation of both light and temperature.

REID, Boston.

PHYSIOLOGY OF THE SPINAL CORD: II. THE INFLUENCE OF CHORDOTOMY ON EXISTING MOTOR DISTURBANCES. OLAN R. HYNDMAN, J. Nerv. & Ment. Dis. **98**:343 (Oct.) 1943.

Hyndman attempted to relieve some of the disagreeable features of motor disturbance by chordotomy in 4 patients. A section of the posterior column had no lasting effect on a patient with severe dystonia musculorum. Only partial diminution of extensor spasticity resulted from anterior chordotomy in a patient with monoplegia in extension of a lower limb due to tumor of the brain. Bilateral anterior chordotomy in a 19 year old youth with spastic tetraplegia and marked clonus resulted in great diminution of the static extensor spasm, but no other change. The same procedure in a patient with pronounced spastic paraplegia in extension due to compression of the cord also caused considerable diminution of extensor spasticity.

A considerable body of experimental evidence is cited in support of the author's observation that preservation of the anterior column, probably the vestibulospinal tract, is necessary for the production of extensor spasticity due to a lesion of the pyramidal tract. Although Tower observed that section of the pyramidal tract alone in the monkey results in flaccid paralysis without hypertonus, hyperreflexia, clonus or a Babinski sign, this condition is attributed to the fact that in animals pyramidal and extrapyramidal fibers are separated in the cord, while in man both types of fibers are incorporated in the pyramidal tract. In the monkey the extrapyramidal system (basal ganglia) is released, while in man the supraspinal system is released after elimination of the pyramidal system.

The author believes that integrity of the anterior column is responsible for the paraplegia in extension which follows a partial lesion of the cord, while the absence of this influence explains the paraplegia in flexion seen after complete section of the cord.

The signs of hyperreflexia in man may have a dual mechanism: (*a*) Integrity of the anterior columns is necessary for acute development of the phenomena, and (*b*) the signs may be of purely spinal origin and require weeks or months for their development. Spasticity is a spinal phenomenon, its only accessory adjunct being the supraspinal influence on the static component of extension. Spasticity and hyperreflexia are separate entities, subserved by different neural mechanisms.

CHODOFF, Langley Field, Va.

MOTOR RESPONSE TO STIMULATION OF CEREBRAL CORTEX IN ABSENCE OF AREAS 4 AND 6 (MACACA MULATTA). M. A. KENNARD and W. S. MCCULLOCH, J. Neurophysiol. **6**:181 (May) 1943.

Kennard and McCulloch stimulated the cerebral cortex of Macaca mulatta monkeys from which both areas 4 and 6 had been removed. Responses shown by the animals which had undergone ablation of the motor areas in infancy differed greatly from those of animals which had been subjected to the same procedure later in life. In the authors' preparations no areas other than those responding to stimulation in normal animals were found to be excitable. Animals from which areas 4 and 6 had been ablated in infancy showed greater excitability of the cortex surrounding the removed areas than did the other animals. Kennard and McCulloch regard the heightened excitability as consistent with the well developed motor performance of the animals, and hence with the functional reorganization within a partially destroyed motor system.

FORSTER, Philadelphia.

EFFECTS OF DORSAL ROOT SECTION ON CHOLINE ESTERASE CONCENTRATION IN SPINAL CORD OF CATS. D. NACHMANSOHN and E. C. HOFF, J. Neurophysiol. **7**:27 (Jan.) 1944.

Nachmansohn and Hoff sectioned extrathecally the dorsal roots of cats between the third and the seventh lumbar vertebra. In some experiments the sections were unilateral and in others bilateral. After operation the animals exhibited the usual signs of dorsal root section. After the animals were killed, the spinal cords from the third to the seventh dorsal roots were removed; the blocks were frozen, and a portion for histologic study was removed from the cephalic and the caudal end of each block. From these portions the percentages of gray and of white matter were determined. From the remaining midportion of the cord the concentration of choline esterase was determined by the Barcroft-Warburg method.

Nachmansohn and Hoff found in normal cats that the choline esterase concentrations for the dorsal quadrants of the cord were 13.7 and 13.9, and those for the ventral quadrants, 17.6 and 18.6. After unilateral differentiation a decrease of 10 to 20 per cent was noted in all four quadrants, and after bilateral differentiation a 30 per cent decrease was observed.

Nachmansohn and Hoff conclude that these experiments offer additional evidence that the role of acetylcholine is the same in both afferent and efferent fibers.

FORSTER, Philadelphia.

CHIMPANZEE FRUSTRATION RESPONSES. G. FINCH, Psychosom. Med. **4**:233 (July) 1942.

Finch attempted to identify the frustration of the chimpanzee and to isolate experimentally the factors which determine the incidence of frustration responses. The experimental part of the investigation was designed to evaluate quantitatively the frustrational significance of variables entering into the frustrating situation. The latter is defined as any situation in which an animal is delayed, hampered, prevented or blocked from reaching a goal object or from beginning, continuing or completing a consummatory act. Observation of various animals in such situations revealed that a chimpanzee's initial response to most frustrating situations apparently involves direct goal seeking. The chimpanzee tries to reach the object with its hands or lips, in spite of an intervening barrier. Failing in such an initial response, he may react by solicitation of help from a human being or another animal, by aggressive reaction, including self aggression, by withdrawal from the frustrating situation, by shift of activity to the search for another object or by expenditure of energy in emotional expression. The observations cited are not mutually exclusive and may occur either successively or simultaneously.

A frustration response is defined as any reaction in which an animal engages after the application of a frustrating situation, but which is not exclusively and immediately directed toward a goal or is not exclusively and immediately directed at resuming the disrupted consummatory act. Experiments were designed which were based on evaluation of the frequency of elicitation of the frustration response. These experiments also enabled a determination of the means whereby changes in the variables affected frustrating situations in which the motivations of hunger drive and food incentive were utilized.

The data obtained in seven experimental situations are described in detail. Throughout the series of tests no attempt was made to evaluate relatively the effects of the various factors being studied. The objectives in the experiments were limited to a demonstration of some of the important variables in a frustrating situation and of the manner in which each variable was related to the incidence of frustration responses. The following observations indicate factors which seem most important in affecting the incidence of frustration responses: (1) A single, isolated frustrating situation was less likely to elicit a frustration response than the same situation preceded by other frustrating situations; (2) when a frustration response occurred, the probability that another frustrating situation presented immediately would evoke another frustration response was less than if the preceding frustration response had not occurred; (3) the incidence of frustration responses increased with (*a*) increased motivation, (*b*) increased expectancy and (*c*) increased conflict; (4) in delayed response problems, frustration response incidence increased as the problem became more difficult.

SCHLEZINGER, Philadelphia.

EAGLE'S FLOCCULATION TEST OF THE SPINAL FLUID. JOÃO BAPTISTA DOS REIS and WALDEMAN CARDOSO, Arq. assist. psicopat. estad. São Paulo **7**:207 (March-June) 1942.

The authors used the Eagle flocculation test in 100 cases of neurosyphilis. In cases of nontreated neurosyphilis the Eagle flocculation and the Wassermann test were equally sensitive. In cases of old, unsuccessfully treated neurosyphilis the Eagle test appeared to be more sensitive than the Wassermann test. In cases in which the disease was under treatment there was a significant parallelism between the Eagle and the Wassermann test. The results of the Wassermann and of the Eagle test were in disagreement in only 7 of 40 cases.

SAVITSKY, New York.

Neuropathology

HYPERTENSIVE DISEASE OF THE BRAIN. I. MARK SCHEINKER, Arch. Path. **36**:289 (Sept.) 1943.

Scheinker describes the characteristic histologic features of hypertensive encephalopathy observed in 25 cases. Typical vascular alterations, confined to the arterioles and capillaries, were observed in all cases and consisted of hyaline degeneration and fibrotic thickening of the walls, associated with narrowing or obliteration of the lumens. The arteriolar changes are considered

a form of arteriolopathy typical of hypertension and differing from the alterations present in the usual case of arteriosclerosis. A working hypothesis advocated by the author presumes that the characteristic arteriolar change may be due to prolonged or repeated functional vascular disturbances.

Changes in the parenchyma of the brain consisted of diffusely scattered, circumscribed small foci of old and recent softening, perivascular hemorrhage, massive hemorrhage and diffuse or localized edema of the brain. The author believes that the alteration of the brain tissue is secondary to the arteriolar lesions.

WINKELMAN, Philadelphia.

OSTEOCHONDROMA OF THE BASE OF THE SKULL. LESTER S. KING and JAMES BUTCHER, Arch. Path. **37**:282 (April) 1944.

King and Butcher point out that four distinct types of cartilaginous tumors can be distinguished: (1) those arising in the paranasal sinuses, with secondary extension into the cranial cavity; (2) those in intimate relation with the meninges, with a probable generic relation to meningioma; (3) those within the brain substance, without evidence of gross attachment to surrounding structures, and (4) chondromatous or osteochondromatous growths arising from the base of the skull.

The case reported by the authors is one of a primary solitary intracranial osteochondroma arising from the base of the skull. The patient, a man aged 24, had had evidence of peptic ulcer for five years. At the time of his last hospitalization he complained of vague headache. Four weeks later he suddenly collapsed and died, with signs of subarachnoid hemorrhage. At autopsy a calcified mass at the base of the skull, previously visualized in a roentgenogram, proved to be a large tumor of the sphenoid bone. It was diagnosed as osteochondroma.

WINKELMAN, Philadelphia.

LABORATORY TRANSMISSION OF ST. LOUIS ENCEPHALITIS VIRUS BY THREE GENERA OF MOSQUITOES. W. M. HAMMON and W. C. REEVES, J. Exper. Med. **78**:241 (Oct.) 1943.

In previous communications Hammon and Reeves outlined the epidemiologic evidence pointing to transmission by mosquitoes of the virus of acute epidemic encephalitis of the St. Louis type. They now report mosquito transmission experiments which were performed in field laboratories in the lower Rio Grande valley, Texas, and in the Yakima valley, Wash. The virus of the St. Louis type of epidemic encephalitis has been successfuly transmitted in the laboratory by the following nine species of mosquitoes, from three genera: Culex tarsalis, Culex pipiens, Culex coronator, Aedes lateralis, Aedes taeniorhynchus, Aedes vexans, Aedes nigromaculis, Theobaldia incidens and Theobaldia inornata. In experiments with Culex tarsalis, infection occurred from the insect's feeding on chickens and ducks which had been previously inoculated by the subcutaneous route. After an incubation period these mosquitoes infected other chickens, and virus was in turn demonstrated in the blood of these fowls. This is interpreted as proof that fowl may serve as reservoirs of virus in nature. Since mosquitoes have been repeatedly observed to be naturally infected with the virus of the St. Louis type of encephalitis and epidemiologic evidence supports their incrimination, their role as vectors is now established.

J. A. M. A.

Psychiatry and Psychopathology

PERSONALITY FACTORS IN PATIENTS WITH MUSCULAR DISABILITY. HERBERT S. RIPLEY, CHARLES BOHNENGEL and ADE T. MILHORAT, Am. J. Psychiat. **99**:781 (May) 1943.

Ripley, Bohnengel and Milhorat studied the personality factors of 67 patients with various types of muscular disease. The adjustment made by the patient was determined in large part by the various psychobiologic factors operating in the formation of his personality makeup, the type of muscular disability, the anatomic distribution, the nature of the onset and the duration and course of the disease. An acute onset of the disability frequently resulted in anxiety, while with a gradual onset gradual adaptation occurred. Resentment toward the affliction was customary at some time or other in the course of the disorder. Psychic trauma was least pronounced in very young patients. The authors found that the type of adjustment depended on a variety of factors. In both diagnosis and treatment, study of personality, as well as physical factors, was found to be valuable.

FORSTER, Philadelphia.

EMOTIONAL FACTORS IN ORGANIC DISEASE OF THE CENTRAL NERVOUS SYSTEM. GEORGE WILSON, CHARLES RUPP and HARVEY BARTLE JR., Am. J. Psychiat. **99**:788 (May) 1943.

Wilson, Rupp and Bartle report a series of cases of organic neurologic disease in which the onset occurred in association with psychic trauma or emotional disturbance. The series includes cases of chronic encephalitis with parkinsonism, multiple sclerosis, pseudosclerosis, dystonia musculorum deformans and cerebral tumor. The authors conclude that emotional trauma may precipitate both physiologic and psychologic alterations.

FORSTER, Philadelphia.

PHYSIOLOGY OF SCHIZOPHRENIC THINKING. GUSTAV BYCHOWSKI, J. Nerv. & Ment. Dis. **98**:368 (Oct.) 1943.

Bychowski discusses the nature of schizophrenic thinking and the linking of the psychologic phenomena with a physiologic substratum. He has noted in schizophrenic thought the presence of phenomena well known to be associated with cerebral lesions, and he believes that in the former there is evidence of functional loss of some cerebral apparatus. Important features are those indicative of a disturbance in categoric function and of agnosia and apraxia of thinking. Thoughts lack direction and centralization, with disintegration of the figures of thought. The waves of energy originating from schizophrenic brains are incomplete and imperfect in regard to tempo and degree of closure. They are not directed by a superior impulse and become independent and unable to reach their goal—hence, the classic symptom of blockage. These phenomena probably originate from subcortical regions, while difficulty in thought and its fractional character may be frontal in origin. The symptom of foreignness of thought, a partial depersonalization, is connected with various parts of the brain, the frontal, parietal and thalamic apparatus chiefly.

Categoric disturbances indicate a dynamic elimination of cortical control with release of primitive function. In schizophrenic patients this is brought about by a lowering of intracortical tension. The functional regression is evidenced by a tendency to the replacement of abstract concepts by concrete images. Disturbances

in the stream of thought indicate dynamic impairment of various cortical and subcortical mechanisms. Apparently heterogeneous, yet basic, schizophrenic symptoms may be localized in an identical physiologic substratum, while a wide variety of symptoms may be based on a varying intensity of the morbid process as localized in different stratums of subcortical and cortical systems. Disturbances in the experiencing of time relations seem to be a basic fault linked with the essential core of the schizophrenic process. This, too, is probably correlated with a dynamic deficiency of cortical and subcortical apparatus.

CHODOFF, Langley Field, Va.

CHARACTEROLOGICAL AND SYMPTOMATIC EXPRESSIONS RELATED TO THE ANAL PHASE OF PSYCHOSEXUAL DEVELOPMENT. WILLIAM C. MENNINGER, Psychoanalyt. Quart. **12**:161, 1943.

Menninger has made an important compilation of the events which occur during the anal phase of psychosexual development and has correlated them with adult expressions related to these infantile experiences. He discusses the events of the anal period under six heads: (1) the stage of sensual pleasure and the initiation of training, (2) the phase of megalomania, (3) the phase of curiosity, (4) the phase of defiance, (5) the phase of sensual pleasure in retention and (6) the phase of giving to gain approval.

He concludes with a consideration of the fate of these six events in adult life; i. e., whether they remain as direct unchanged primitive survivals, as socially unacceptable character traits, as socially acceptable character traits, as reaction formations or as symptoms. He considers, also, the complicated relation of the oral, anal and genital phases of psychosexual development. He questions whether the primal scene is really the first object of the child's curiosity. He believes that the child is curious as to the parents' eating and as to excretory acts before he becomes interested in the primal scene, and he believes, also, that some of the fear he shows about it may be a carry-over of something he has already experienced during the anal stage.

The article is a valuable summary of knowledge of the anal stage and its end results.

PEARSON, Philadelphia.

EFFORT INTOLERANCE IN SOLDIERS. MAXWELL JONES and R. SCARISBRICK, War Med. **2**:901 (Nov.) 1942.

Jones and Scarisbrick report on the study and treatment of 500 soldiers with the effort syndrome. They believe the condition is essentially a neurosis and have classified their patients on the basis of effort response, anxiety and vegetative lability, as follows:

A. Poor effort response from an early age without any anxiety directed to the body.

1. A history of poor effort response but no history or signs of excessive vegetative lability and no anxiety about the poor physical performance. There was 1 instance of such a condition among the 500 patients examined.

2. A history of vegetative lability and a poor effort response but no concern or anxiety about the autonomic dysfunction (3 per cent).

3. Chronic anxiety states with anxiety not directed to the body (9 per cent). The patients usually have poor effort tolerance.

B. Poor effort response manifested later in life without anxiety directed to the body.

4. Adequate physical and psychologic makeup, with development in adult life, for no apparent reason, of symptoms of vegetative dysequilibrium, with some decrease in physical performance but no anxiety in relation to the symptoms (1 per cent).

4 (*a*). Sinus and paroxysmal tachycardias with no disproportionate anxiety about the symptoms and admission of little, or no, physical disability (2 per cent).

5. Acute anxiety states with somatic symptoms accompanied by objectless fear or fear of harm not connected with the body and little effort intolerance (9 per cent).

C. Poor effort response with anxiety directed to some part of the body.

6. A history of vegetative lability from earliest childhood, at first unaccompanied by anxiety, with later development of anxiety in connection with some part of the body, usually the heart (24 per cent). Their effort response is poor.

7. Chronic anxiety states like those listed in paragraph 3, but the anxiety is directed to some part of the body (28 per cent). The effort tolerance is poor.

8. No apparent physical or psychologic predisposition (24 per cent). The anxiety is associated with a particular organ. Effort intolerance is the rule.

Several tests may be used in determining effort intolerance. The exercise tolerance test has a limited value because it cannot be divorced from emotional reactions.

Since breathlessness is the most common complaint (93 per cent of patients), since the mean volume per breath is only 520 cc. for patients with effort intolerance, as compared with 910 cc. for control subjects, and since the average respiratory rate for patients with effort intolerance is 20.5, as compared with 11.7 for control subjects, the respiratory exercise tolerance test is important.

In maximum effort tests exhaustion occurred in men with effort intolerance in an average of two minutes, while of the control subjects 6 were exhausted in six to ten minutes and 7 could continue indefinitely without exhaustion.

Determinations of the lactic acid content and the p_H of the blood are not reliable because patients with effort intolerance break down before the evidences of physiologic fatigue appear in the blood.

The authors' method of treatment is as follows:

On admission the patient is tested psychometrically and for vocational ability. He is then placed under a strict routine and given such work as the tests indicate will be most suitable. Attendance at educational lectures, discussion groups and organized activities is compulsory, as is graduated physical exercise.

The main difficulties in treatment lie in the patient's ideas of his symptoms and in the absence of a desire to get well. For these difficulties there are group discussions on medical and psychologic topics three times a week. The authors have found that such group discussions are more valuable for inhibited persons than medical discussions with the physician.

The nurses are trained to undertake the individual treatment of patients.

PEARSON, Philadelphia.

NEUROSES IN SOLDIERS: USE OF SODIUM AMYTAL AS AN AID TO PSYCHOTHERAPY. LEON L. ALTMAN, War Med. **3**:267 (March) 1943.

Altman reports a series of cases of neuroses in soldiers in which he used sodium amytal as an adjuvant measure to psychotherapy. The use of the drug is preceded, accompanied and followed by investigation, explanation, positive suggestion and reassurance, and is

valuable in demonstrating to the patient that his disability is not irreversible. The author discusses at some length the role played by suggestion in the origin and treatment of hysterical disabilities.

PEARSON, Philadelphia.

MILD DEPRESSIVE PSYCHOSIS. B. ANDRATSCHKE and C. H. ROGERSON, Brit. M. J. **1**:780 (June 26) 1943.

Rogerson states that depressive states probably occur more frequently than is generally realized. In defining depression, he quotes Lewis: "Any person who is unhappy and ill with his unhappiness may properly be said to be in a state of depression." Because Rogerson believes that in many cases the condition is recognized much too late to be treated successfully, he has made a study of 100 consecutive patients with mild psychotic depression, in an attempt to familiarize others with the clinical picture which this condition produces. In his series there was no predisposition to sex, nor was there any occupational bias. The majority of patients were between the ages of 20 and 60. Almost all the patients admitted feeling depressed; 78 per cent complained chiefly of physical symptoms, and many had complaints of "fear," "anxiety," "agitation" and "inadequacy." The previous personality was characterized by overconscientiousness, pessimism or cheerfulness. Precipitating factors, particularly psychologic, but occasionally physical, were noted in 82 per cent of patients. Approximately half the patients had had one or more previous attacks of depression. Sixty-two of 83 patients gave a family history of a psychologic abnormality. Rogerson states that remission of untreated depressions is gradual. However, mild depressions can be relieved in the majority of patients by the induction of three to eight convulsions. He emphasizes the importance of treating the psychologic precipitating factor once the depression has been overcome.

ECHOLS, New Orleans.

Vegetative and Endocrine Systems

EFFECTS OF LOW ATMOSPHERIC PRESSURES ON THE ACTIVITY OF THE THYROID, REPRODUCTIVE SYSTEM AND ANTERIOR LOBE OF THE PITUITARY IN THE RAT. ALBERT S. GORDON, FRANK J. TORNETTA, SAVINA A. D'ANGELO and HARRY A. CHARIPPER, Endocrinology **33**:366 (Dec.) 1943.

Adult male rats subjected to atmospheric pressures of 280 to 250 mm. of mercury for six hours daily for fourteen to twenty days showed an increase in weight of the adrenals and a decrease in weight of the testes, the seminal vesicles, the ventral prostate, the thymus and, possibly, the thyroid. The weight of the pituitary gland was not affected. The loss in body weight was 8.7 per cent. Male rats subjected to the same atmospheric pressures for eighteen to twenty hours daily for varying periods showed an increase in weight of the adrenals and a decrease in weight of the testes which were less than those occurring in the rats exposed to low pressure for shorter periods. The decrease in testicular weight was due mainly to degeneration of the spermatogenic tissue; slight atrophy of the interstitial tissue occurred. Injections of large doses of chorionic gonadotropin did not repair the spermatogenic tissue but caused pronounced hypertrophy of the interstitial tissue.

Failure of the male gonad under the low atmospheric pressure conditions of the experiment was reflected in an increased potency in gonadotropic principle of the anterior lobe of the hypophysis. The number of basophils increased, and castration-like cells appeared in the pituitary gland of such animals.

Low pressures produced little effect on the thyroid.

The morphologic and physiologic changes in the anterior lobe of the pituitary were probably a reflection of the effects of the reduced pressures exerted directly on other endocrine glands, such as the testes and possibly the thyroid; however, the possibility of a direct influence of the low pressures on the tropic mechanisms of the pituitary gland could not be excluded.

PALMER, Philadelphia.

DIFFERENTIAL CONCENTRATION OF HORMONES IN THE CENTRAL AND PERIPHERAL ZONES OF THE BOVINE ANTERIOR PITUITARY GLAND. GEORGE K. SMELSER, Endocrinology **34**:39 (Jan.) 1944.

Smelser found that in the anterior lobe of the beef pituitary gland the thyrotropic, the adrenotropic and the gonadotropic principles were all present in greater concentration in extracts of tissue taken from the basophilic central zone than in the more peripheral, and predominantly acidophilic and chromophobic cortical, portion. Although the ratio of basophilic material contained in any preparation of central and peripheral tissue was fixed, the concentrations of the aforementioned principles in the center and at the periphery were not the same.

The author suggests, therefore, that the thyrotropic, adrenotropic and gonadotropic principles of the beef pituitary gland are produced or stored by several distinct cell types, which have different spatial distributions in the gland.

PALMER, Philadelphia.

PUBERTAS PRAECOX IN A FEMALE INFANT CAUSED BY VENTRICULAR CYST. DAVID J. FLICKER, J. Nerv. & Ment. Dis. **98**:42 (July) 1943.

Flicker adds a fourth confirmed case to the 3 previously reported instances of pubertas precox in females. The case was that of a girl who was first seen at the age of 3 years because of precocious sexual development and failing eyesight. In addition, there were spastic hemiplegia and hyperalgesia on the right side, primary atrophy of both optic nerves and nystagmus on looking to the right. Encephalographic examination revealed enlargement of both lateral ventricles, the left being the larger; ventriculographic studies showed a small, circumscribed spherical mass in the region of the pineal gland. A frontoparietotemporal flap was laid down, and a cyst of the third ventricle was encountered.

CHODOFF, Langley Field, Va.

Treatment, Neurosurgery

AGRANULOCYTOSIS FOLLOWING MALARIAL THERAPY IN GENERAL PARESIS. T. J. HELDT and G. A. GODER, J. Nerv. & Ment. Dis. **98**:248 (Sept.) 1943.

Heldt and Goder mention the possible complications of malaria therapy in treatment of dementia paralytica and report a case of agranulocytosis. Four similar cases have been reported, with 1 fatality. Their case is that of a white man aged 45 with dementia paralytica who was treated with benign tertian malaria, with the induction of twelve chills in fifteen days. After this, an intravenous injection of 25,000,000 killed typhoid organisms was given, with a subsequent rise in temperature to 106 F. After induction of two more chills, it was decided to terminate the malaria therapy, and 10 grains (0.65 Gm.) of quinine was given and the dose repeated in three hours. However, the temperature

remained elevated and increasing cyanosis and dyspnea developed. At this time a blood count revealed only 751 white cells, none of which were of the granulocyte series. The patient died the next day. Autopsy revealed dementia paralytica, lobar pneumonia, old subdural hematoma, splenomegaly and hepatomegaly, and hemangioma of the liver. An idiosyncrasy to quinine is considered, but is not believed likely.

CHODOFF, Langley Field, Va.

THE CONSERVATIVE APPROACH TO THE USE OF SHOCK THERAPY IN MENTAL ILLNESS. S. D. ROSEN, L. SECUNDA and K. H. FINLEY, Psychiatric Quart. **17**:617 (Oct.) 1943.

Rosen, Secunda and Finley advocate a "middle of the road" policy in the choice of shock therapy. They state that it is best not to give shock therapy in cases in which there is a history of lifelong maladjustment or in which recovery through psychotherapy alone is likely in one or two months. They prefer metrazol as the shock agent if curare or beta-erythroidine hydrochloride is used to modify the convulsions. Limitation and spacing of treatments are both important. They point out that patients may be given treatments in too rapid succession, without allowance of time for improvement before the next, and perhaps damaging, shock. Shock therapy is only a means of making the patient more accessible so that psychotherapy may be used.

Fourteen cases were studied in most of which the psychosis was predominantly depressive. In no case were more than seven treatments given. In no case did electroencephalographic studies reveal permanent changes in the cortical rhythm after convulsive therapy. Clinical improvement could not be correlated with the electroencephalogram.

McCARTER, Philadelphia.

SULFADIAZINE THERAPY OF PURULENT MENINGITIS. HARRY A. FELDMAN, LEWIS K. SWEET and HARRY F. DOWLING, War Med. **2**:995 (Nov.) 1942.

The authors report the results of treatment with sulfadiazine in a series of 42 consecutive cases of purulent meningitis.

Of 24 patients with meningococcic meningitis, ranging in age from 10 months to 57 years, 22 recovered without complications.

Four of 5 patients with meningitis caused by Haemophilus influenzae type B and treated by sulfadiazine and rabbit serum recovered. None of the 6 patients with pneumococcic meningitis recovered.

The authors conclude that sulfadiazine gives excellent results when used alone in the treatment of meningococcic meningitis. In cases of meningitis caused by type B H. influenzae and the pneumococcus, specific serum should be given in addition to the sulfadiazine.

PEARSON, Philadelphia.

PLANNING FOR THE TREATMENT OF HEAD INJURIES. H. CAIRNS, Brit. M. J. **1**:313 (March 13) 1943.

Cairns offers information which should assist in the formulation of plans for postwar treatment of injuries of the head. Although "the late results of blunt head injuries have been painted blacker than they really are," statistics are cited to show that the majority of patients are able to resume normal activity comparatively early. Whereas most injuries to the head can be treated by a trained accident surgeon in the hospital, the complicated cases should be reserved for the neurosurgeon. There is a need for more precise statistics regarding the number, type and distribution of accidents than are now available. The accident surgeon should have a clear understanding of the problems of treatment and rehabilitation of patients with head injuries. Institutional rehabilitation should be required only for patients with severe or recalcitrant injury and for those whose home conditions are unfavorable.

ECHOLS, New Orleans.

Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY, AND NEW YORK NEUROLOGICAL SOCIETY

GERALD R. JAMEISON, M.D., *Chairman, Section of Neurology and Psychiatry, Presiding*
Joint Meeting, Dec. 14, 1943

The Type of Soldier Who Succeeds. COLONEL WILLIAM C. PORTER, Medical Corps, Army of the United States.

The honeymoon stage of Selective Service terminated when the United States entered the war and when it was necessary to make this country the arsenal of democracy and, at the same time, to build up an army of 10,000,000 men to carry out the military program. The high rate both of rejection and of discharge for neuropsychiatric disorders, which have simultaneously increased to unprecedented levels, cannot continue in the face of conservation of man power. A more positive attitude toward selection and toward retention of certain military personnel of substandard mental levels must be substituted for the discredited experiment of making a better army by the simple method of more rejections and more medical discharges. Soldiers must be selected and retained on the basis of positive attributes and of the something they can contribute to the military effort.

A number of cases of mental deviants who have been successful in the service are described. It is not what one has, it is what one does with what one has, that counts.

DISCUSSION

DR. LAWRENCE S. KUBIE: It is a privilege to discuss Colonel Porter's paper. The essence of his argument, however, should not be misunderstood. Colonel Porter does not argue that standards must be relaxed because an occasional good soldier is lost among those who are rejected on psychiatric grounds. Safety and common sense indicate that until fewer errors can be made, the mistakes would better be on the side of too many rejections. The lesson Colonel Porter draws from these exceptional cases is that the basic military norms have never been clearly established. On this all must agree, and it is on this point that I shall offer a few suggestions.

In view of this lack of any clear standard of military normality, it is not surprising that the rate of discharge from the Army among troops inducted through stations with a high rate of rejection is just as high as, and in some instances even higher than, the rate of discharge from the Army among soldiers inducted through stations with a low rate of rejection. This means that the induction boards do not know what to accept and what to reject. In medicine the definition of normality (which in military medicine means the condition necessary for a soldier to succeed) is always more difficult than the definition of a pathologic deviation. Such a definition of military normality is lacking because there is no permanent "setup" in any branch of the armed forces the sole task of which it is to study the problem. This is a defect in organization, and technical procedures will never be developed to select the potentially successful soldier and to reject the potential failure until there are a number of basic improvements in the organization by which men are chosen for the services.

The necessary organizational changes are simple and obvious. First, selection must be in the hands of a permanent organization in the armed forces the task of which is selection, and nothing but selection, twenty-four hours a day, three hundred and sixty-five days a year, in peacetime, as well as in wartime. It must be possible for a man to make a career in the art of selection, because selection is a specialty in its own right. It is not the practice of medicine, and never will be. In the armed forces, as they are set up today, there is no provision for such an agency. The medical aspects of selection are conducted by physicians who are assigned to that task only temporarily. It is not a task which they relish; and in peacetime the physician works at odds with the recruiting officer, just as he works at cross purposes with the draft board in wartime. The recruiting officer is rewarded on the basis of the number of men he gets into the services, and he is never penalized for his mistakes. The physician may strive for quality, but the recruiting officer wants quantity. No one has presented the difficulties of that task better than General Lull, in an article published in the *Military Surgeon* (**72**:297 [April] 1933). He pointed out that about 70 per cent of all enlisted men discharged for disability over a twenty year period of peace had been eliminated for conditions which had existed prior to their enlistment. It should be borne in mind that this occurred during peacetime, while there was all the time in the world! Sixty per cent of these men were discharged within a year after enlistment, and about 12 per cent within the first month of service. A large majority had neuropsychiatric conditions. If peacetime procedures are no more competent than this, how is it possible to do better during the rush and urgency of war? It is true, of course, that the man power which is drawn into the armed services in times of peace does not constitute a true cross section of the country. It is a selected group, not selected for favorable personality traits, either. This fact has further implications for the problem under consideration. Whether or not the citizens of this country like the idea, there probably will never be a first class job of selection until there is universal compulsory military training. Only then will the physician, the psychologist and the classification officer have an opportunity to work with a representative cross section of the man power of the country.

A permanent selective service organ of the armed forces would have to be medically manned. That does not mean that it should be under the Surgeon General's Office. The Surgeon General and his entire staff are physicians who practice medicine in the Army. Their thoughts are on the problems of sanitation and health. During times of peace the problems of selection drop completely out of sight. That is proved by a review of the literature, that is, of the articles published by members of the medical services of the Army and Navy during times of peace. Immediately after a war, for a year or so, a few interesting studies of the problems of

selection appear in the various journals of military medicine. Within a year or so, however, such articles disappear, and there are almost none until the next war breaks out. This indicates how completely the Medical Department of the Army turns its thoughts away from this basic problem. It cannot be permitted to happen again, for the major function of a small peacetime army or navy is to be prepared to expand.

The most important single agency for rapid expansion is the nonexistent permanent selective service organ of the army. In providing for the existence of such an organ of selection, many technical problems must be considered. I shall refer to only one. There was a day when every man had a gun and knew how to use it. His gun hung on his wall, and he became a soldier by taking the gun off the wall and joining some of his neighbors as they marched down the lane. There was no induction, no selection, no classification. There was no training period because there was nothing he had to be taught how to do. He became a soldier solely by placing himself under a system of command. Armies have now grown to millions. Training takes months or years. Yet the old tradition still dominates all military organizations. With this growth, with the mechanization of transportation and of firearms, it became no longer possible for an army to forage its way along. Thus, the service of supplies grew in importance until it finally became one of the basic elements of the Army, and now is called the Army Service Forces, on a par with the Army Ground Forces and the Army Air Forces. This was a necessary change; unfortunately, as the service grew, it became a catch-all within which practically all functions are dumped which are not specifically those of the Army Air Forces or the Army Ground Forces. The Surgeon General's Office, and all other vital personnel, are lower in command, way down the line in the Army Service Forces. Consequently, the forces concerned with the task of selection and rejection, of classification and transfer and promotion, of rehabilitation and discharge, are scattered and subdivided and subordinated to the authority of a service with which they should be at least coordinate. The personnel staff is without the authority which it needs in order to do the job it has to do. Any one who has studied this problem carefully realizes that no advance in technical procedures, however great, will bring a solution until the Army personnel force is created on a par with the Army Air Forces, the Army Ground Forces and the Army Service Forces.

Furthermore, the entering soldier should be under the authority of the Army personnel forces until he is classified and is through his basic training. This division should be the primary school of the soldier. Then he should be delivered over to the line command for definitive combat training and for duty with combat units, only to be returned to the Army personnel forces when he is ready for rehabilitation and for discharge.

When the Army has such a system as this, it will be in a position to bring to bear all that is known on the problem of selection. Even more important, it will have an organization which can conduct the investigations and make the observations during times of peace, as well as of war, without which the questions which Colonel Porter has raised will never be answered.

Therapeutic and Psychosomatic Aspects of War Neuroses. DR. PAUL H. HOCH (by invitation).

The etiologic factors in war neuroses and the treatment employed are discussed. In the series of patients under treatment narcosuggestive or narcocathartic forms of therapy were used chiefly. Sodium amytal was injected intravenously until the patient was in a state of drowsiness, and then the choice depending on the mental state, suggestion or catharsis was employed. The results were uniformly satisfactory, and about 75 per cent of the patients so treated recovered.

An objective method of examination of the patients with war neuroses was tried. It was found that the psychogalvanometer is a useful instrument in the demonstration of tension. A large number of patients with war neuroses were examined and compared with patients who were free from such disorders. The patients were exposed to a sound picture which reproduced the effect of torpedoing. The emotional tension during and after exposure to the picture was recorded. In normal persons the tension after seeing the picture died down rather quickly, while in the patients with war neuroses the tension persisted. It was possible, also, to demonstrate the efficacy of certain therapeutic methods by this form of registering tension.

DISCUSSION

DR. CLIFFORD D. MOORE, Newtown, Conn. (by invitation): I regret that Dr. Hoch was unable to read the part of his paper which referred to causation, because it is a clearcut presentation of a great deal of material that has been recorded on the subject. However, to come to that part of the paper which has been read, the use of the motion picture to reproduce the type of disaster that led to the traumatic war neurosis and the employment of this mechanical device to measure tension are noteworthy. For a long time this question of the measurement of tension, related as it is to anxiety, has been in the minds of investigators. The procedure Dr. Hoch described now offers one a means of measuring the tension, of evaluating the progress of patients under treatment and of comparing the results of the various therapeutic methods that have been employed for traumatic war neuroses and for neuroses occurring in civilian life. However, my main concern at the moment is the aftermath of the neurosis—the social, medical and general economic problem of the postwar period. The figures on the incidence of neuropsychiatric casualties in the armed forces—and it should be remembered that the merchant seaman, under consideration here, is not a member of the armed forces; he is a member of an industrial group whose counterpart may be found in any of the industries familiar in peacetime—indicate that there will be continuing neuroses not only in men who have broken under the stress and strain of war, whether a seaman or a member of the armed forces, but also in persons who fail to adjust in the transition from war service to peacetime living.

Another part of Dr. Hoch's paper which was not read referred to chronicity. The question arises whether the men who are being seen, not for the first time at the rest center, but after an interval, for the second or third time, will eventually, either in wartime or in the postwar period, continue to have a neurosis, or, indeed, whether the so-called neuroses of the patients who are now being seen may not develop in the postwar period into frank psychosis. All that has been said in the literature on the treatment of war neuroses has, I think, a special interest for physicians who will be engaged in civilian hospital practice. By the employment of the method which Dr. Hoch has outlined, the supportive, suggestive group therapeutic approach will be one of the advances that comes out of the present struggle.

Experiments in the Rehabilitation of Discharged Service Men. DR. THOMAS C. A. RENNIE (by invitation).

DISCUSSION

DR. J. H. W. VAN OPHUIJSEN: As is known, the idea of the rehabilitation clinic was conceived by Dr. Rennie. During the summer I had a conference with Dr. Rennie in which he asked me to start a clinic similar to the one he had planned to open at the New York Hospital, and I was only too glad to follow his example. The rehabilitation clinic at Lenox Hill Hospital was officially opened on Oct. 7, 1943. It has been active for a much shorter period than the clinic at the New York Hospital, and it operates on a much smaller scale. Instead of ten psychiatrists, there are four. The number may increase shortly to six. There are five social service workers, two psychologists and one secretary. All the persons who cooperate in this plan are volunteers, as no funds are available for the project. The procedure followed by the clinic is exactly the same as that employed in Dr. Rennie's clinic. The patients are seen by appointment; each patient is interviewed first by a social worker, then by a psychiatrist and then, if necessary, by a psychologist, who gives him an intelligence test or a Rorschach test; finally, the whole group decides on what shall be done for the patient. There has been contact with many agencies.

It is gratifying to be able to state that many of the percentages which Dr. Rennie gave correspond with those which my associates and I have obtained thus far, though with a much smaller number of patients. The New York Hospital clinic registered 128 patients from August 19 to the time of writing. The Lenox Hill Hospital clinic, only 41. Twenty per cent of the patients who registered at the New York Hospital clinic did not come for the initial appointment, and 22 per cent of the patients who registered at the Lenox Hill Hospital failed to do so. So 102 patients were seen at the New York Hospital clinic and 32 patients at the Lenox Hill Hospital clinic. The figures for rejected men and for men discharged from the Army were 20 and 80 per cent respectively for the New York Hospital and 19 and 81 per cent respectively for the Lenox Hill Hospital. The largest group of patients was that of men between the ages of 20 and 30—constituting 64 per cent of the patients at the New York Hospital clinic and 56 per cent at the Lenox Hill Hospital clinic. I do not have a figure on the number of patients from the New York Hospital clinic who were unemployed. Our figure was 30 per cent, and a number of these patients were unemployed by choice, I should say. They were waiting for an opportunity to get a job which they liked; the number would certainly have been smaller if they had followed the example of the majority and had taken the jobs which they could easily have had. The similarity of the aforementioned figures seems to indicate that the groups of patients, although small, are representative of the total.

There is one notable difference between the way in which the two clinics have carried out their work. With the enormous number of rejected and discharged persons in the New York area, a great demand for treatment was anticipated. It must be admitted that the demand was not great, in the case either of the New York Hospital clinic or of the Lenox Hill Hospital clinic—only 128 out of over 90,000 persons in the area in three and a half months. At any rate, it was expected that a large number of requests for treatment would be received and that the clinics would never be able to handle all the material. My associates and I tried, therefore, to find a criterion by which we could choose the patients for treatment in our clinic. We did not know the exact numbers of rejected and discharged men, although we could have obtained those figures. We did not know anything about the geographic distribution of the men, the distribution according to local draft boards or the distribution with regard to the nature and the duration of the psychiatric disturbances. So we decided we would see as many patients as we could and examine them as thoroughly as we could, but keep for treatment only those whose illness had developed lately in connection with the threat of induction, with the disappointment of rejection or with military service. The choice of patients according to this criterion would make our treatment rehabilitation in the true sense of the word. The other patients would be referred to other clinics.

To our surprise, we found that the number of patients with "fresh" illness was exceedingly small. Of 32 patients, 87 per cent stated that their illness was of long duration, and when I say "long" I mean "many years," "since childhood," "ten years ago," "five years ago," even "eighteen years ago." In only 4 patients did the disorder become apparent during military service, and in none did nervous or psychotic symptoms develop in reaction to the threat of induction or in reaction to rejection. Four patients, that is, 12.5 per cent only, had recently become ill, and they are being treated at the clinic. Moreover, only 9 per cent of the patients with a long-standing psychiatric disorder had ever sought medical treatment (9 per cent of Dr. Rennie's patients had consulted a private psychiatrist).

When these facts are considered together—(1) the surprisingly small demand for treatment, (2) the surprisingly large number of patients with psychiatric disorders of long standing and (3) the striking absence of treatment in the past—one comes to the conclusion that there is little opportunity for rehabilitation in this group, but that it represents many more persons who are mentally ill than we expected, just as we found many more than the expected number of persons with disorders of the ear, eye, heart and teeth. All these patients with psychiatric conditions are in need of treatment at some time or other. It is doubtful whether rehabilitation clinics could provide them with the necessary facilities for treatment.

DR. RICHARD BRICKNER: Dr. Rennie has done a pioneer work, not only in having set going the active effort to rehabilitate some of the men discharged from the Army but in helping to bring to the attention the appalling number of people in this country who need neuropsychiatric help. It seems at times as though almost half the population would have to be trained in psychiatry to take care of themselves and the other half. Cobb has given a recent estimate of over 6,000,000 people in the civilian population who are known to be in need of and are receiving care, and this is a conservative figure. It is important, in the treatment of some of the men who are being discharged from the Army, to realize that they might have continued in civilian life with the adequate adjustment which they had been making prior to induction had they not been placed in a situation to which they could not adjust.

The immense problem now before psychiatrists cannot possibly be met adequately by any clinical method now available; it calls for the development of new, rapid

methods of procedure and for new and enlarged technics of group therapy—technics probably not yet conceived of. Attention should be directed toward these goals promptly. Beginnings have, indeed, been made.

It is interesting to review some of the clinically new situations. I can refer to them better in terms of rejection than of discharge because most of my own experience has been with the induction boards. Rejection by the Army may precipitate a latent neurotic state, which may take one of several forms: (1) severe guilt feelings, the rejected man believing that civilian clothes advertise what is the matter with him, and (2) the attitude that society as a whole has rejected him and that he has no usefulness to the world. In my experience this type of patient profits particularly from quick psychotherapy, which can even be given in the interview at which the man is rejected. The mere fact that the physician himself accepts the registrant as a man can have considerable therapeutic value, especially if a few minutes is given to interpretation of the meaning of rejection. It should be explained that rejection does not signify entire lack of value to society, but that it merely indicates that the registrant is so constructed that he would probably not make a good soldier, although he might serve society excellently in other ways. Such a conversation, which states a simple truth in a welcome way, frequently produces visible relief.

The attitude of many laymen, employers and girls in particular, toward rejected and discharged service men has significance. The present experience may yield some profit in the education lay persons are receiving about the nature of psychoneurotic states. I am sure the educational measures now being taken can accomplish something. I overheard a girl talking to another in the theater district one night. She said, "I heard on the radio these 4F's are not all so bad; perhaps we had better pay more attention to them." Many employers have no comprehension of the real values involved, but many others have or are learning about it, profitably, for the first time.

I should like to raise a question suggested by Colonel Porter's paper and ask him one. He commented on the magnitude of the psychoneurotic situation. He suggested that if the rejection rate of candidates for the services should reach 25 per cent, it might imply the decadence of civilization. The war experience and the magnificent way in which most of the men are carrying it through show that people are anything but decadent, at least under conditions of emergency. Of course, under ordinary circumstances the behavior of wartime is not expected, and should not be. But that so many people become mentally ill merely by living in the present day civilization suggests, not decadence, but a defect in the arrangement of society. This problem should have a great deal of consideration by members in the medical profession; it should not be left entirely to specialists in politics and economics, who cannot have the same experience in human woe that is open to neuropsychiatrists.

Colonel Porter aroused an anxiety among medical examiners, without dispelling it. It is recognized that there must be psychoneurotic persons who do well in the Army. We examiners reject them, I think, too much by rote, and we have always been worried about it; there are certain classes of persons whom the Army says it does not want, and that is all given by rule. There is no follow-up system, either, for checking our mistakes. Colonel Porter indicated in general terms how the examinations might be improved; I wonder if he could formulate that material more specifically.

Colonel William C. Porter, Washington, D. C.: I must confess my failure to do more than state, in the twenty-five minutes allotted me, that most persons have potentialities or assets that counterbalance many of the traits which might be listed as liabilities. The assessment of these positive traits should be the concern of the medical officer, rather than assessment exclusively of the liabilities.

Dr. Thomas C. A. Rennie: Two men out of the 100 patients at our clinic could have gone back into service, and 3 others, if they had been handled differently, might perhaps have stayed in the service. Perhaps 5 per cent of the disabled men might have made good soldiers. Does Colonel Porter feel there are more than 5 per cent?

The problem of rehabilitation is here, and it must be handled some time. I wish to point out that there is a new federal program; a new law has been passed, the Barden Bill, Public Law 113, which permits the inclusion of the psychiatrically handicapped in a federal plan of rehabilitation. It will become imperative to see that the psychiatric casualties are included in this federal plan. Within the week I have spoken to a representative of the National Selective Service about the establishment of a national program of rehabilitation. This coming week I am to see a representative of the United States Public Health Service who is anxious to see a program of psychiatric rehabilitation established. The Veterans Administration wishes to do its part, and there will probably be something of a scramble before it is decided who is going to have the responsibility for the psychiatric casualties of this war.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

William G. Lennox, M.D., *Presiding*

Regular Meeting, Dec. 14, 1943

Pressure Lesions in Nerve. Dr. D. Denny-Brown and Dr. Charles Brenner.

The material of this paper was published as two articles in the Archives (Paralysis of Nerve Induced by Direct Pressure and by Tourniquet, **51**:1 [Jan.]; Lesion in Peripheral Nerve Resulting from Compression by Spring Clip, **52**:1 [July] 1944).

Effect of War on the Civilian Population in England. Dr. Walter S. Maclay, London.

England entered the war with over 3,000 hospitals, of which 2,000 were administered by local authorities under the supervision of the Ministry of Health, but were largely uncoordinated, while the remaining 1,000 were self-governing institutions. It was clear that to deal efficiently with war casualties and with illness among the evacuees and to coordinate the civilian and military needs a unified medical service would be required. In 1939 the Emergency Medical Service was established as a division of the Ministry of Health for this purpose.

In 1938 there were 370,000 hospital beds, of which 130,000 were in hospitals for mental disease. The government estimated that 300,000 beds should be made available, and these were provided by restriction of

admissions, by introduction of extra beds into existing hospital premises and by a building program of huts as annexes.

The large hospitals in the cities were the places most likely to be bombed; so they had to be used as casualty clearing hospitals, while hospitals in the country were used as base hospitals. This has led to an interesting development in hospital administration, since the skilled medical staffs which formerly tended to be concentrated in the large teaching centers and hospitals in the cities are now distributed throughout the country.

Specialist centers had to be provided for treatment of orthopedic conditions, burns, head injuries, cutaneous diseases, etc., and these were placed in general hospitals. Centers for treatment of neuroses were also required, and these, like the others, were established chiefly in general hospitals. The hospital of which I am in charge is the largest of the centers for the care of patients with neuroses and is placed near the outskirts of London. It cares for both civilian and service patients, of both sexes. The following comments apply only to civilians and refer to attempts made by various workers to determine the effects of bombing and evacuation.

Effect of War on Chronic Neurotic Patients.—At the outbreak of war the heavy air raids did not materialize, and it was difficult to be certain what effect fear and anticipation would have on the mental health of the civilian population. The subject was complicated further by the lack of knowledge about the frequency of minor neurotic illnesses under ordinary circumstances and by the vast migrations of the population, as a result not only of evacuation but of dispersal of industry and administration.

My colleagues and I examined 100 patients with chronic neuroses who had been in regular attendance at outpatient clinics of London hospitals for at least one year prior to the war. More than half, mostly patients with obsessions, hypochondriacal conditions and hysteria, were not more in need of medical attention then than they were under normal circumstances. Over a quarter of the patients were adversely affected in that more acute symptoms had developed. They were chiefly patients suffering from chronic anxiety states and depression.

Effect of Bombing.—The crude figures for attendance at and admission to hospitals suggested a considerable reduction of the incidence of neuroses and psychoses except in two groups of patients: In general there was an increase in the rate of admission of patients over 75 years of age and of mentally defective persons. Most of the patients suffering from acute shock due to air raids recovered within a few days, and, of those that did not, another group recovered within five weeks. There were, however, a certain number who, although they did not require hospital care, continued over prolonged periods to receive allowances for disability. In order to assess the severity of their illness and to prevent or cut short the course of neuroses with compensation claims, the Ministry of Pensions offered these patients admission to a neurosis center.

Ministry of Pensions Patients with Prolonged Neurosis.—The cases of the first 100 of the aforementioned patients admitted to the hospital were reviewed. The results were sufficiently satisfactory to suggest adoption of the same plan of hospitalization for other patients with neurotic illness complicated by pension or compensation claims. Although a number of unsuitable patients were included, e. g., those with arteriosclerosis and schizophrenia, 76 per cent of the patients treated returned to work of some kind, usually in about three months, whereas the average period of disability prior to admission was eight months. In an analysis of the cases it was found that numerous factors, many of them unrelated to war, contributed to the prolongation of the neurotic reaction to bombing.

Minor Effects and After-Effects.—One of my colleagues had the opportunity to interview and examine all the patients with no physical injury admitted during a certain period to a first aid post at one of the most heavily bombed places in England, where at some time or other every second inhabitant had passed through a first aid post. The investigation was carried out two months after the air raid experience. At that time two thirds of the persons involved had no neurotic symptoms; half of this number had never had any symptoms, and the other half had had temporary nervous symptoms, from which they had recovered during the intervening months. One third of the total number still had symptoms at the time of the investigation. The type of neurosis among persons who had been personally involved was predominantly anxiety or depression or a combination of the two. Previous unstable personality is often responsible for the persistence of the neurosis, but continuous residence in the danger area and abnormal living conditions also make for chronicity, and these factors should be eliminated if possible.

Children.—It is well known that the effect of bombing on children has not given rise to any serious problem. Children appear to react in the same way as do the adults with whom they are associated. The problems of evacuation have been much greater with respect both to young children, who suffer by sudden separation from their parents, and to older children, among whom there has been an increase in delinquency, apparently due in large part to disruption of family life.

Industry.—In 1942 the loss of working weeks as a result of sickness was such that about 800,000 extra people had to be maintained to make up the loss. These would formerly have been drawn from the 2,000,000 unemployed, but this group is no longer available. This loss of time represents a great increase over the figures for 1939 and suggests that the problem of war weariness requires attention.

DISCUSSION

DR. HARRY C. SOLOMON: Will Dr. Maclay state why he thinks there is such a change in the incidence of the effort syndrome and whether he feels that anxiety has replaced all other forms of neurotic behavior?

DR. WALTER S. MACLAY, London, England: The effort syndrome was a matter for concern at the beginning of the war. A factor in the apparent decrease in the incidence of the effort syndrome was the instruction issued to the regimental medical officers that they were to examine the patient and then make up their minds, without dithering about, and this helped prevent the development of the effort syndrome. Again, this syndrome was called by many other names. As examining physicians, we were told, "Now, don't confuse the patients with effort syndrome and the patients with real neuroses." We would say, "No," and put the diagnosis of effort syndrome on the record and the real diagnosis in brackets. Dr. Paul Wood and Dr. Adolph Abrahams were working with us at the time, and they swung over to the point of view of psychiatry, or of psychosomatic medicine, as Wood called it. He swung too far over. We began to have trouble in making

proper psychiatric diagnoses; so we had to divide the patients into three groups: (1) patients with a constitutional basis and effort intolerance; (2) patients with a constitutional basis and a neurotic outlook toward that tendency (these patients had an embryonic neurosis before the war, and symptoms developed according to the personality, so that there was a wide variety of diagnoses) and (3) patients in whom psychogenic factors were predominant.

In looking for objective signs by which to measure the degree of fatigue, we found that the most useful method was determination of the pulse rate and the lactate content of the blood. With the first group we used the bicycle ergometer and worked the patients to the point of exhaustion, in about twenty minutes. We found that the lactate content of the blood had risen to over 100 mg. per hundred cubic centimeters. The second group was exhausted in ten minutes, and the lactate level rose to 100 mg. The third group gave up, and the lactate content of the blood was 30 or 40 mg. per hundred cubic centimeters.

Dr. Solomon's second question about anxiety was occasioned by a wrong statement on my part. In the hospital we use the term "anxiety state" because the patients describe anxiety as the presenting symptom. Anxiety is the commonest condition, but depression and obsession are nearly as common.

DR. ABRAHAM MYERSON: Is there any relation between the lowered health level and poor diet or lack of vitamins?

DR. WALTER S. MACLAY, London, England: Rationing is one of the government's outstanding accomplishments. People have had to learn different ways of cooking and to use new foods, and the general health has been improved by the war diet. I was alarmed when all the soldier patients lost weight during their first week in the hospital and all the civilians gained weight. However, I believe there is no deficiency in the diet.

DR. ABRAHAM MYERSON: Is there any increase in alcoholism?

DR. WALTER S. MACLAY, London, England: No, because there is not enough alcohol.

DR. ALEXANDRA ADLER: Is there any reason that neuroses developed in some previously normal soldiers and did not appear in other, equally normal, ones?

DR. WALTER S. MACLAY, London, England: I cannot answer that. I think that under sufficient stress a neurosis will develop in any one. But why it occurs in some persons and not in others depends on the fact that the stress differs for different people.

CAPTAIN A. WARREN STEARNS: Can you pick out in advance the patients who will have neuroses?

DR. WALTER S. MACLAY, London, England: I have found that the induction boards are sworn at in this country, too. In England psychiatrists are not used on the induction boards. The boards used to have psychiatric consultants to whom they could send patients with doubtful conditions, but they found that with such patients the psychiatrist always said, "Dont use this man"; so after a while they did not bother even to refer him.

DR. HENRY R. VIETS: Dr. Maclay spoke of hysteria. I have gained the impression from the literature that there is not a great deal of hysteria in this war. It was supposed to be common in the last war. Is it true that gross hysteria is less common in the present war?

DR. WALTER S. MACLAY, London, England: This statement is not true. We see a great deal of hysteria. The nearer one is to the front, the more gross hysteria and conversion one sees. We have a unit of fifty beds for these patients. and it is always full. The chief types are amnesia and stammering; there are hysterical blindness and deafness, disordered gaits and paralysis. There were many cases of hysteria after Dunkerque.

DR. HENRY R. VIETS: Do you treat persons with acute hysteria by the continuous narcosis method?

DR. WALTER S. MACLAY: We now have a small number of patients whom we treat with narcosis, but we had most of them after Dunkerque. We give 2 cc. of somnifene (diethyl-diallylbarbiturate of diethylamine) and then sodium amytal to make them sleep twenty hours out of the twenty-four. It seems to me that in this country physicians use such deep narcosis that the patients are completely toxic, and I cannot see that the results are any better. We next give the patient insulin. We give it in the morning and get him back to his occupational work in the afternoon. In the case of the hysterical patient we try to remove the symptom with narcoanalysis, and for this we give sodium pentothal. For amnesia we give sodium amytal. It restores lost memory. We do not like hypnosis.

WILLIAM G. LENNOX, M.D., *Presiding*

Regular Meeting, Jan. 20, 1944

Role of Trauma in the Causation of Amyotrophic Lateral Sclerosis. DR. PAUL B. JOSSMANN.

The role of trauma in the causation of amyotrophic lateral sclerosis has been discussed for many decades. Charcot himself mentioned trauma as a possible etiologic factor. Since the specific causative agent of amyotrophic lateral sclerosis is unknown, the significance of trauma is still a controversial issue. At present the enormous increase in traumatic injuries to soldiers, as well as to civilian workers, not only provides an unusual opportunity for further studies but raises many practical questions of medicolegal character. Because of the theoretic and practical implications of the problems, it seems justifiable to present a relatively large material of 66 cases of clinically verified amyotrophic lateral sclerosis. In 7 of these cases there was a history of trauma preceding the disease. In analysis of the material the following desiderata and criteria were tested: (1) statistical evidence, (2) clinical evidence and (3) anatomic evidence. The percentages as reported in the literature differ widely. They obviously depend on the selection of cases and on various accidental factors. Interestingly, the incidence of amyotrophic lateral sclerosis during the first world war did not exceed the peacetime figure. Jeliffe's paper (*J. Nerv. & Ment. Dis.* **82**:415 [Oct.]; 532 [Nov.] 1935) comprehends 90 some cases which were reported in the literature since about 1860. This number is not impressive in view of the total incidence of amyotrophic lateral sclerosis and serves as a warning against uncritical reliance on the mere statistical evidence. Clinical evidence has been based on the presence of two criteria: First, trauma should precede the onset of amyotrophic lateral sclerosis, and, second, the injured part should present the first neurologic signs. Both desiderata are controversial issues, especially with regard to the relatively poor knowledge of the chronology of the disease and its pathology. Frequently it is impossible to determine what is cause and what is effect. There is no scientific

explanation of whether or how a peripheral lesion can initiate a systemic disease of the spinal cord. There is a possibility that the injured part may be more susceptible to an otherwise independent process. It is also possible that the injured part simply displays the neurologic signs more obviously. A closer analysis of the material along the critical lines indicated left only 2 cases which fulfil the criteria. Therefore the clinical evidence cannot be considered a wholly reliable basis for a decision as to the causal significance of trauma. As for the anatomic evidence, the postmortem observations are not helpful in solution of the problem. They simply confirm the clinical diagnosis, without supplying evidence of the causal relation. The paper by Waggoner and Löwenberg (Role of Trauma in Amyotrophic Lateral Sclerosis, ARCH. NEUROL. & PSYCHIAT. **45**:296 [Feb.] 1941) provides crucial evidence that traumatic lesions and a systemic process can coexist without any interdependence. In summary, no scientific basis could be demonstrated on which one may rely in deciding on the causal relation between trauma and amyotrophic lateral sclerosis. There is no evidence that trauma can cause amyotrophic lateral sclerosis. The term "precipitant" may serve the medicolegal needs in decisions in cases in which the absence of a "somehow contributing" traumatic influence cannot be proved.

DISCUSSION

DR. HENRY R. VIETS: I highly approve of Dr. Jossmann's conservative attitude. I say this particularly because no neurologic disease seems to have been more bandied about in the last ten years than this syndrome. The reasons for this are well known in this country: A few years ago a noted athlete died of the disease, with a good deal of publicity, and about the same time vitamin E was suggested as a possible cure.

In his original article, Charcot described an intrinsic, degenerative disease, beginning in the cervical region, to which he gave the queer name amyotrophic lateral sclerosis, the term combining a clinical sign and a pathologic lesion. The disease was always symmetric, beginning in the small muscles of the hands and progressing to the legs. One still sees examples of this pure type, but the disease as Charcot described it is rare.

When my colleagues and I were investigating the effect of vitamin E on this disease at the Massachusetts General Hospital a few years ago, we studied many cases to find a few in which the condition fitted Charcot's description. The others were atypical; in some cases there were bulbar lesions; in others the disease began with unilateral signs, and in still others lesions appeared first in the lower part of the spinal cord. This mixed group of cases was not easy to classify, but only those in which the disease picture was reasonably close to that of Charcot's syndrome were included under amyotrophic lateral sclerosis. The disease is not easy to diagnose, and I judge from Dr. Jossmann's tables that he, too, had difficulties. Some of his cases, at least, do not fit into a rigid classification.

If one should see a patient who was traumatized in the lowest cervical region and within six months or a year presented the signs of amyotrophic lateral sclerosis, one might consider it good evidence of a causal relation between trauma and amyotrophic lateral sclerosis. Dr. Jossmann's conclusions, in my experience, are justified.

Late Effects of Birth Injuries. DR. CLEMENS E. BENDA, Wrentham, Mass.

The late effects of birth injuries are still the subject of much controversy. In order to obtain suitable material, the selection of cases must be made with the application of strict criteria.

In my autopsy material of 125 cases of various types of mental deficiency, the pathologic picture in 24 cases was considered the result of a birth trauma. In 5 cases there was subcortical encephalomalacia or granular atrophy, and in 3 cases, central encephalomalacia or cystic degeneration; in 16 cases widespread lesions involved the cortex and the basal ganglia.

In addition to the pathologic changes, the clinical aspects confirmed by autopsy are discussed.

DISCUSSION

DR. BRONSON CROTHERS: I congratulate Dr. Benda on a fine piece of work. The evidence he had gathered accords with my observations. It makes sense to refer birth injuries to the mechanism of birth if possible. My associates and I have had several cases of erythroblastoma in which the cause was not a birth injury. In these cases signs of neurologic damage developed within three or four days and persisted. This is an important disease, and the problem has not yet been given fair attention.

Of interest to me is the management of and prognosis for children with birth injuries. I have taken care of so many such children, and for so long, that I feel that I have some notion of the abysmal failure in their management thus far. With good handling, a child with cerebral palsy gets along well until adolescence. Then, some of them discover that everything that they have been told is false. They have been assured by every one that if they will do their exercises and muscle training faithfully, they will be worth something. In the end they may not be able to make a living. Within two months I have received frantic letters from 4 patients who went through good standard colleges and now cannot get jobs. These young people are from families who can furnish a fair amount of protection for them, but in the end it will be withdrawn. The theory of enthusiasts is that no one need despair because he has an athetoid condition. Dr. Elizabeth Lord had begun to formulate the opinion that if the child had intrinsic charm and an adequate income, there was no reason that he should not thrive. Guaranteed the intrinsic gift for adjusting well socially, the patient with cerebral palsy gets along fairly well. This does not apply to the child with simple hemiplegia, but to the athetoid child. My colleagues and I now have records for a large number of children extending over a period of years.

My plea is for realistic appraisal and reappraisal of the future of these children and for a ruthless review of the advice which we as physicians give them. Until something can be done to open opportunities for self maintenance as adults, these children and their parents are being permitted to live in an unreal world.

DR. PAUL I. YAKOVLEV, Waverley, Mass.: One is impressed by the thoroughness of documentation with which Dr. Benda has presented his argument. There are several points in his paper which inspire thought and which I wish to accent. Dr. Benda's classification of the late pathologic features of birth injury as defined by him is useful. He divides his material into three groups: (1) cases of diffuse mantle sclerosis, (2) cases of circumscribed porencephalic defects and (3) cases of central thrombophlebitic necrosis in the plane of the striothalamic sulcus. I am intrigued by the high incidence of epilepsy in cases of the last type. The necrotic lesions here occur in the morphogenetically important

zone of the brain—the zone of cleavage between the hemispheres and the brain stem. The analogy of the localization of necrotizing lesions in these cases with the localization of the morphogenetically determined lesions in the same zone in cases of epilepsy associated with tuberous sclerosis is at once apparent and, in my opinion, is significant for the high incidence of epilepsy in cases of lesions due to birth injury.

The clinical aspects of the cerebral lesions occurring at birth, in infancy or in early childhood is another interesting point in Dr. Benda's paper. It is significant that in describing the symptoms of infantile cerebral lesions and in appraising their pathophysiologic basis, so distinct from the symptoms of similar lesions in the adult and the pathophysiologic factors involved in their production, one often has to speak in terms of "extrapyramidal" disorders. This invites an explanation: The reason for the clinical differences between the infantile cerebral lesions and those occurring in adult life apparently lies in the fact that the old, "polyneuronic," tegmental motor system in the newborn is still bare, i. e., not yet invested by the new, "oligoneuronic" projection system from the cerebral cortex to the final common path. The frequency and apparently great facility with which infantile cerebral lesions lead to a syndrome of decerebrate rigidity is an illustration in point. It is known that in lower mammals, such as the dog or cat, simple decortication is not sufficient to produce decerebrate rigidity. One has to shear off a whole hemisphere and a good part of the brain stem at the level of the colliculi to produce the syndrome. Not so in man. As a result of a far more advanced encephalization of functions of the nervous system in man, a lesion of the cortex alone, or of the frontal lobe alone, is often sufficient to cause the state of decerebration, and this is especially so in an infant, for in the newborn human infant the tegmental (old) motor system is wholly divested of the cortical control of the proprioceptive function and remains so for several months after birth. It is understandable, then, that a lesion of such a highly encephalized organism as the human, at a time when all the proprioceptive functions are still in a state of mere anticipation of their eventual cortical organization, should lead to decerebration with particular ease. This is supported by daily clinical experience. Thus, I have seen cases, and Dr. Benda showed one, illustrating how a focal and unilateral lesion at birth may lead to the state of lasting bilateral decerebration. This brings me to the last point, viz., that so far as the clinical implications, prognosis and treatment are concerned, the significance of all congenital and infantile lesions lies not so much in the site or extent or nature of the pathologic condition of the brain as in the degree and the manner in which such a condition, occurring at a critical period of development, distorts the future organization of the functions of the nervous system.

Dr. Paul B. Jossmann: Did Dr. Benda have any evidence in his cases of increased intracranial pressure, such as choking of the disks, and did he make air studies? In 1928 I published a series of 4 cases, all of children with cystic malformations in the brain. All were admitted because of suspected tumor of the brain. One child was so ill that he was subjected to air studies, and a diagnosis of porencephalic cystic formation was made during his lifetime. The roentgenograms showed, first, an entirely unconnected ventricular system and, second, a system of various large and small cysts, which were interconnected. The horizontal section of the brain shown in Dr. Benda's second group of cases resembled the picture in this case. The two pictures are almost alike. In some cases it might be possible through air studies to make the diagnosis during the patient's lifetime. This paper has not merely academic interest but practical implications as well.

Dr. Louis Goodman, Howard, R. I.: Dr. Benda's demonstration has been convincing. However, only in 1 case was the possibility of an antecedent inflammatory lesion mentioned.

I have done autopsies in 3 cases in which the history of an acute inflammatory process was associated with the development of the neurologic symptoms. These 3 cases showed a remarkable similarity to some in Dr. Benda's second group. In the first case there is said to have been an attack of poliomyelitis at the age of 1 year, after which weakness developed on the right side. The child presented a severe behavior problem and was mentally retarded. In the second case there was a history of epileptic seizures from the age of 18 months, associated with hemiparesis. Mention is made in the history of the possibility of antecedent anterior poliomyelitis. In the third case, the child, at the age of 7 months, is said to have awakened one night screaming, and the next morning she was found to have left hemiplegia and a speech defect. Two of these patients died at the age of 38, and the other, at the age of 59 years.

In each case the lesion was unilateral, as it was in some of the sections which Dr. Benda showed. No evidence of an active inflammatory lesion was present, and the histopathologic picture was one of degeneration, reminiscent in 1 case of the picture in polyserositis (Pick's disease).

In view of the clinical history in each of these cases, which indicated either an infectious or an inflammatory origin of the lesions, rather than a birth injury, I should like Dr. Benda to comment on this aspect of the problem.

Dr. Raymond Morrison, Boston: Dr. Benda has pointed out, in this excellent paper, the large, dense internal capsule in 1 of the cases of his first group. This point needs some explanation, which I am sure Dr. Benda can give. If this case was one of birth injury, in which the cortex was practically destroyed on the left side, how does it happen that the internal capsule could develop and become medullated at all? On the other hand, since, obviousy, the internal capsule did become medullated and the patient lived for thirteen years with an atrophic cortex, how is it that the internal capsule did not degenerate?

Status marmoratus is a condition spoken of a great deal and seen but seldom. The section shown here illustrated the lesion well, although usually it is accompanied by an abundance of very small fibers, which unite, as tributaries, to form larger, denser bands, as shown by Dr. Benda. In cases in which this occurs, there is, with the overgrowth of myelin, a hyperplasia not only of the accompanying oligodendrocytes but, I believe, of nerve cells. Dr. Benda has speculated about asphyxia's being a possible cause of status marmoratus; it happens, however, that oligodendrocytes and nerve cells are the elements in the central nervous system most susceptible to lack of oxygen. It does not seem likely, therefore, that under conditions of anoxia these cells would become hyperplastic.

Dr. Madelaine R. Brown: In 2 of Dr. Benda's cases birth was by cesarean section. How does birth

injury occur during such an operation? In which group do these cases fall?

DR. WILLIAM G. LENNOX: Birth injury, which is of etiologic significance in later life, may not show itself early. At least this seems to be true of epilepsy. In a series of nearly 2,000 patients with seizures, a history of difficult or instrumental birth was obtained several times as frequently as in a normal control group.

DR. CLEMENS E. BENDA, Wrentham, Mass.: Dr. Crothers has always emphasized the importance of birth injuries. When I first approached this problem, seven years ago, there was a general feeling that in most cases mental deficiency was due to a congenital malformation, and that even when a birth injury had occurred, it was not the cause of the deficiency. It is a point of great satisfaction to me that my results agree so well with Dr. Crothers' observations and that I am now able to demonstrate the importance of birth injuries on the basis of an extensive pathologic material.

The management of children with birth injuries is outside my field. I hoped that a neurosurgeon could suggest something. Treatment of birth injuries should begin right after birth, not five or ten years later. The mortality would be great, but some measure should be tried. I think the outcome would be different.

As to Dr. Yakovlev's discussion of epilepsy: The relation of this disease to birth injury presents a difficult problem. I have not made up my mind entirely. It is impressive to a pathologist to see how epilepsy may occur with lesions of every type. Under the influence of Penfield, one feels that cortical lesions are of major importance. If one studies the whole brain, however, one finds that the lesions are so widespread that the surgical approach could be of no great help. Dr. Yakovlev's idea that epilepsy is a "central" disturbance may be supported by the appearance of epileptic seizures right after birth, when the cortex is not yet fully developed. How can a day old child go into status epilepticus if epilepsy has a cortical origin?

As to Dr. Jossmann's question about an increase in intracranial pressure: The children are not seen at the time when the intracranial pressure can be checked, but autopsy shows compression and swelling of the brain. Sometimes the cortical convolutions are adherent to each other. There must have been a period of increased intracranial pressure, cerebral swelling and optic nerve atrophy. Many of the children in my series had previously been subjected to air studies in the Children's Hospital; these studies revealed bilateral asymmetric enlargement of the ventricles and considerable air over the cortex. On none of these children had air studies been made recently; if they had, it would have been easy to demonstrate cystic degeneration.

With regard to Dr. Goodman's question: This material is made up of cases in which there was evidence that no other factor was present. I have cases in which an inflammatory process produced, for example, mental degeneration or cortical atrophy. I do not think the pictures in my cases are characteristic of birth injury, but they may be produced by trauma at birth, if no other factors are at hand.

With regard to Dr. Morrison's question: In the section which I presented the internal capsule contained chiefly fibers coming from the frontal and precentral lobes, which were not destroyed in that case. In more caudal sections the internal capsule showed some reduction, but its development indicated that it had previously been normal. One may conclude, therefore, that the damage to the brain occurred after these structures were laid out; in other words, the lesion was not a congenital one, which developed before birth. In my opinion, the lesion had its beginning at the time of birth but progressed slowly, as a degenerative process.

As for status marmoratus, Dr. Alexander expressed the belief that the condition is a congenital malformation which developed before birth and is distinguishable from status fibrosis, but he did not consider the recent literature on this subject. As a result of several recent studies of status marmoratus, the condition is regarded not as a malformation but as a lesion which develops after birth, a kind of central "neurinoma," similar to the neurinoma following amputation.

With regard to Dr. Brown's question about cesarean section, two factors seem to contribute to birth injuries. Usually the cesarean section is made, as a last resort, after the child is asphyxiated. Rapid, precipitate birth seems to produce lesions of the same type as does slow birth. The process is similar to that in a diver coming up to the surface too quickly. The vessels are congested, and a sudden release of pressure causes hemorrhage.

In both cases in my series cesarean section was elective and was done when the mother was in good condition. On the basis of the cerebral lesions, both cases belonged in group 3. There were generalized atrophy and asymmetry of the brain, as well as areas of demyelination and fibrosis throughout the cortex and the basal ganglia.

In answer to Dr. Lennox: Can one recognize birth injuries at birth or not? In the majority of my cases signs appeared shortly after birth, and I did not include all cases in which mental defect developed later in life. Epileptic seizures in the first year frequently indicate birth injury, but epilepsy developing in later life is not so certainly due to such an injury.

CHICAGO NEUROLOGICAL SOCIETY

R. P. MACKAY, M.D., *President, in the Chair*

Regular Meeting, Dec. 14, 1943

Pharmacologic Aspects of Synaptic Depression in The Autonomic and Central Nervous Systems. DR. AMEDEO S. MARRAZZI (by invitation).

Neurosurgery in the European Theater of War. LIEUTENANT COLONEL LOYAL DAVIS.

Hourglass Neurinoma of the Right Vagus Nerve. DR. PERCIVAL BAILEY and (by invitation) DR. MANUEL SANCHEZ.

Suppression of Motor Responses in Man. LIEUTENANT HUGH W. GAROL, Medical Corps, Army of the United States (by invitation) and DR. PAUL C. BUCY.

This article appeared in full in the June 1944 issue of the ARCHIVES, page 528.

R. P. MACKAY, M.D., *President, in the Chair*

Regular Meeting, Jan. 11, 1944

Neuromyelitis Optica: Report of a Case. DR. JOSEPH P. REICH (by invitation).

A man aged 35 became ill with a "cold," apparently accompanied by sinusitis; at the same time loss of vision occurred in the right eye, resulting in complete blindness within two days; there were also weakness and paresthesia in both lower extremities.

Examination revealed complete absence of vision in the right eye, with papilledema and small hemorrhages; the left eye was normal. There were no other neurologic signs except for absence of the abdominal cutaneous reflexes. Examination of the blood and urine showed nothing abnormal. The total protein of the cerebrospinal fluid measured 40 mg. per hundred cubic centimeters; there were no other changes. On the following day there were hypalgesia and hypesthesia for vibratory stimuli below the twelfth dorsal segment, as well as a suggestion of the Babinski sign on the right side and of the Rossolimo reflex on the left. Within the next week there developed flaccid paraplegia, with paralysis of the abdominal muscles, retention of urine and a sensory loss below the level of the lowest ribs. The paralysis progressed to complete quadriplegia, and death occurred suddenly, four weeks after the onset of the disease, apparently as the result of paralysis of the muscles of respiration.

The general pathoanatomic changes were as follows: cloudy swelling of the myocardium and the liver, acute hyperplasia of the spleen, edema and partial collapse of the lungs, acute hemorrhagic cystitis and retroperitoneal lymphosarcoma anterior to the aorta. Inspection of the brain revealed hyperemia of the subarachnoid vessels; microscopic examination showed moderate hyperemia, with no cellular infiltration. The spinal cord showed severe hyperemia of the subarachnoid vessels, with complete disappearance of the normal texture, as revealed in a large number of sections. The gray and the white substance could not be differentiated. The most severe changes were noted in the lumbar region, with a few foci of hemorrhages in the lowermost portion. Microscopic study showed destruction of the nerve tissue, with *Gitterzellen* containing either foamy cytoplasm or fat droplets. Some of the smaller blood vessels were surrounded by lymphocytes, monocytes and occasional polymorphonuclear leukocytes; some contained marginal thrombi. The myelin sheaths had undergone advanced degeneration. The axis-cylinders were damaged in places. The right optic nerve showed demyelination and absence of axis-cylinders, with slight infiltration of lymphocytes in the septums.

From a diagnostic point of view, the absence of disseminated patches and the outspoken involvement of the gray substance differentiated the condition in this case from multiple sclerosis. More difficult was the differentiation from encephalomyelitis. The literature contains descriptions of an almost pure degenerative process on one side of the brain, of outspoken inflammatory changes on the other and of all degrees of transitional states. Furthermore, there is the contemporaneous occurrence of a considerable number of cases of encephalomyelitis, of optic neuritis with signs of involvement of the cerebrum or the cord and of isolated optic neuritis.

I have encountered 3 cases of optic neuritis with rapidly developing blindness and 1 case of myelitis almost contemporaneous with the case reported here, an occurrence which may have been more than a mere coincidence. Some of the anatomic changes in this case, and in other cases, resemble the lesions encountered in poliomyelitis. Changes in the optic nerve occur occasionally in the course of this disease. In an epidemic in Silesia, Germany, in 1929, Guttmann observed 5 cases of neuritis optica among 22 cases of poliomyelitis.

The clinical and anatomic observations justify the question whether optic neuromyelitis is a definitive nosologic entity or whether it should be considered as a link in a chain of toxi-infectious diseases which present the various reaction types, degeneration, inflammation and vascular changes, in different degree.

Prolapsed Cervical Intervertebral Disks. DR. ADRIEN VERBRUGGHEN.

It is only in recent years that the syndrome of herniation of the nucleus pulposus of a cervical intervertebral disk has been partially understood. Before this there were many sporadic references to lesions in the neck which probably originated in the disks. These lesions, however, were treated surgically on the assumption that they were tumors of the spinal cord, which in fact they were, for they caused compression of the spinal cord of varying degrees. However, the herniations which protrude laterally and only slightly into the cervical portion of the spinal canal, and which cause only root pain, were not fully described until publication of the article of Semmes and Murphey (The Syndrome of Unilateral Rupture of the Sixth Cervical Intervertebral Disk, J. A. M. A. **121**:1209 [April 10] 1943). These authors described a typical syndrome of root pain extending down the arm with hypesthesia of the index finger, as well as precordial pain and extension of pain down the back to the vertebral border of the scapula. This syndrome was usually associated with stiff neck and pain on coughing and sneezing.

It is to this syndrome in particular that I wish to refer. I have encountered 3 proved cases of root pain due to herniation of a disk in the cervical region and 1 case in which a more serious protrusion caused paraplegia, with a Brown-Séquard syndrome. In the last case operation was performed by my associate Captain C. S. Textor II, at Cook County Hospital, in the belief that the lesion was a tumor of the spinal cord. He removed transdurally a protruded disk, which in former days would probably have been diagnosed as chondroma.

In only 1 of the 3 cases in which there were typical root pain, precordial pain, extending to the vertebral border of the scapula and stiff neck did the typical changes described by Semmes and Murphey appear. In the first case operation was performed at Presbyterian Hospital, on April 15, 1943, exactly a week after the article of Semmes and Murphey was published. The patient presented the typical picture of numbness of the index finger and pain extending down the arm. In the other 2 cases, however, there was complete absence of objective neurologic signs, and the only reason the complaints were taken seriously was the discrete, precise location of the pain. Previously, in both cases the diagnosis either of neurosis or of malingering had been made by other physicians, and in both cases it was fortunate that the roentgenograms showed slight abnormalities which I felt justified exploration for a possible herniated disk. Fortunately, in both cases, the disk was removed, and the operation was followed by complete and uneventful recovery. In 1 case slight numbness

in the index finger, which had not been there before, developed after operation. The pain in the arm, however, was gone.

The question of which nerve root is involved is difficult to decide, and the various descriptions of the dermatomes in the hands differ somewhat. Semmes and Murphey stated the belief that the index finger in their cases was supplied by the seventh cervical root. In the first case in which I operated, I am not sure which root was relieved by removal of the herniation, but if any positive statement were to be made, I should say it was the sixth cervical. In the last case, that of the patient who had numbness in his index finger after the operation, I am certain from postoperative roentgenograms that the nerve root involved was the sixth cervical. In the second case the fifth cervical nerve root was involved; that could be stated from evidence both in the preoperative and in the postoperative roentgenograms. These 3 cases, then, seem to cast doubt either on the accuracy of the statement of Semmes and Murphey, that the seventh cervical root was involved, or on my own, that the sixth cervical root was involved.

Another curious phenomenon was the postaxial distribution of pain. Patients have pain down the back of the arm and down the back of the vertebral border of the scapula. It may be true in the cervical region, as it seems to be in the lumbar region, that only the postaxial part of the nerve root is involved in compression of the disk. No explanation can be offered for this at present.

It appears, then, that careful observation will have to be made on patients with such herniations and their persistent root pain, for by no means all with herniated cervical intervertebral disks present the typical symptom complex described by Semmes and Murphey.

The Frontal Lobes and the Higher Levels of Consciousness. DR. WARD C. HALSTEAD (by invitation).

In this paper are reported some of the residual neuropsychologic observations on a patient studied over a period of five and one-half years following partial removal of the left frontal lobe. An American-born man aged 48, healthy and with an intelligence quotient of 110, was operated on by Dr. Percival Bailey at the University of Chicago Clinics in 1935. After his death by accident, five and a half years later, a complete autopsy report and the files of the interested insurance companies were made available to me for study. Serial sections of both hemispheres of the brain were prepared for microscopic study. Because of the length of time during which this patient was under investigation, it was possible to differentiate clearly between transitory and residual alterations or deviations from normal performance in his postoperative behavior. With the exception of residual bilateral anosmia, repeated postoperative neurologic examinations revealed no clinical signs of an intracranial lesion. There was no history of postoperative convulsions, and no abnormalities were noted in repeated electroencephalographic recordings. At no time could aphasia, specific agnosias or apraxias be demonstrated.

Evidence of an alteration in grouping behavior in this patient has been presented elsewhere (Halstead, W.: *Am. J. Psychiat.* **96**:1263, 1940 [case 1]. Halstead, W., and Settlage, P. H.: Grouping Behavior of Normal Persons and of Persons with Lesions of the Brain, ARCH. NEUROL. & PSYCHIAT. **49**:489 [April] 1943). Brightness discrimination was found to be disturbed beyond normal limits in a situation in which voluntary movements of the eyes of increasing magnitude were imposed as part of the task. The normally present size-weight illusion was observed to be absent when studies were made by means of appropriate test objects. In contrast to simple reaction time, which was normal, the reaction time required for choice between a light and a sound was abnormally lengthened on the average and was extremely variable. Critical fusion frequency was lowered throughout 3 logarithm units of intensity, the greatest lowering being apparent at low intensities. The variability in successive determinations was unusually low. Tactile sensitivity, as measured by the von Frey hairs, was normal for the volar surfaces of the forearms. For a given examination, the thresholds were fairly stable and were quite similar in the two arms. The visual fields, as explored by standard methods of perimetry and campimetry, were normally full. Notable constriction of the dynamic visual field was found, however, for targets exposed briefly and simultaneously at the fovea and at the periphery. This constriction of the dynamic visual field varied systematically with the projection distance, a characteristic which differentiated it from tunnel vision. A similar constriction has been found to result from frontal lobotomy. These various changes represent an alteration in the higher levels of consciousness.

DISCUSSION

DR. WILLIAM WINDLE: Dr. Halstead has compared the performance of a patient with the mean of performance of a normal, or control group. I wonder whether he would say more about this control group.

DR. WALTER FREEMAN, Washington, D. C.: Dr. Halstead, I believe, is thinking along the lines of the mental energy factor; so the dynamic forces constitute the important part of the situation. In the patient he has described a certain defect was produced by operation, which in certain test situations made him less efficient than normal, but in other test situations, such as determinations of weight, made him more efficient. In other words, the patient was unable to gather together the various impressions from the different fields of afferent stimuli in order to produce a response which was equivalent to the average, or normal, response.

Dr. Halstead has presented a number of tests, which he will admit are very much of the laboratory type, and are not particularly related to real life. I should like to ask under what circumstances the patient met his death, and whether the accident was due to his being insufficiently protected by the neural mechanism of a proper escape reaction possessed by the normal person. Dr. Halstead mentioned the emotional aspect of the situation only briefly, in what he calls the catastrophic reaction. I refer to the test in which the patient would try to solve only 6 of 9 items. In the other tests Dr. Halstead described, was the patient in harmonious working order, or did he present a certain lackadaisical attitude toward the tests, which interfered with adequate performance? I ask that particularly with respect to determinations of the dynamic visual field, which is a test of extraordinary importance for the study of the physiology of the higher functions of the cortex.

DR. WARD C. HALSTEAD: The problem of adequate controls, raised by Dr. Windle, becomes complex in the field of behavior. I presented the curve for the performance of the brain-injured patient in the category tests, as compared with that for normal controls, as a convenient means of illustrating the variability of the

patient's performance with respect to these simple test items. The average deviation of the small group of normal subjects, deliberately selected to include children and adults with different degrees of intelligence and formal educational background, was shown by the shaded zone surrounding the line representing the mean values.

In answer to Dr. Freeman, I believe there is an important energy factor—which is not yet sufficiently well understood to be termed "mental"—in the behavior of the brain-injured person. This factor is not adequately reflected, for example, by the standardized tests for intelligence. I believe it is related to vigilance in the sense in which Head used the term. As suggested in my paper, it is believed that critical fusion frequency may provide a quantitative method for dealing with this factor. Studies along this line have been under way in my laboratory for some time.

The death of this patient resulted from a fall on an icy sidewalk. After careful investigation, the interested insurance companies paid claims for accidental death involving a considerable sum of money.

Time did not permit an adequate presentation of the emotional responses of this patient. I may state that he was highly cooperative in the attitude which he adopted and maintained toward the many laboratory situations in which he was placed. After his discharge from the hospital, he never once missed or was late for an appointment to come back to the laboratory.

I agree with Dr. Freeman that the situations which I described this evening are definitely "on the laboratory side." I cannot agree, however, that they are "not particularly related to real life"; I believe there is not yet sufficient basis for an evaluation of the practicability of these tests.

Book Reviews

The War and Mental Health in England. By James W. Mackintosh, M.D. Price, $0.85. Pp. 91. New York: The Commonwealth Fund; London: Oxford University Press, 1944.

Dr. Mackintosh, professor of preventive medicine at the University of Glasgow, in ninety-one pages, gives a clear, informative picture of the mental health in England during the first four years of the present war and offers suggestions with respect to mental hygiene for the remainder of the war and for the postwar period.

The book is divided into two parts: "The Impact of War" and "Mobilization for Peace." In the first part, the author devotes one chapter to each of the first four years of the war, and he emphasizes the specific problems for each year. Some of his material dealing with juvenile delinquency and the problems of evacuated children, their parents and foster parents is not new to the readers of current medical literature. Much of this material corroborates what has already been described by Martha Elliot, Susan Isaacs, Anna Freud, Dorothy Burlingham and others. One wishes that the author had given more space to the problems of mental hygiene of the adult. For example, he mentions only in passing the problems of old people who were not evacuated and the emotional problem of the young wife who "has not the moral character to live a celibate existence while surrounded by the pick of the Allied forces, all suffering from homesickness. The sometimes inarticulate British husband away in the Middle East suffers acute mental distress over his wife's attitude and particularly the effect on his children."

The author briefly describes the existence of a "shelter neurosis," which is "represented by an unreasoning urge to take shelter on every possible occasion," and he points out that claustrophobia was a comparatively rare condition.

In the second part of the book, the author discusses the care of patients with mental disorders before the outbreak of war and the use of mental disease hospitals for surgical cases, and the like, during the war and points out that, although these hospitals were overcrowded, there was a reduction in the number of commitments for mental disorders.

In this section of the book, the author describes the present training of psychiatrists and psychiatric social workers and states that the training of social workers "tends to be too intensive," whereas "the teaching of psychiatry to undergraduates is not at present given its proper place in the medical curriculum. The subject is not presented to the student in its true perspective in relation to clinical medicine and is apt to be dismissed as a short extramural course consisting of a few lectures with the minimum of practical clinical study." The postgraduate training of psychiatrists is divided into two parts: the one for persons who intend to practice in mental disease hospitals, and the other—a higher qualification—for those who wish to enter the rank of "outside" consultants. The author points out that at present a great deal of thought is being given in England to the teaching of preventive and social medicine and that surveys are under way in England and Scotland to consider the existing psychiatric services and the question of teaching. To quote again: "Both committees have had some difficulty in adjusting the conflicting claims of the neurologist and the psychiatrist."

Dr. Mackintosh discusses other mental hygiene problems of the future and stresses education as the central feature in the effort to prevent, rather than treat, emotional disorders.

The book, although touching every problem only superficially, has value and is recommended to all interested in problems of postwar mental hygiene.